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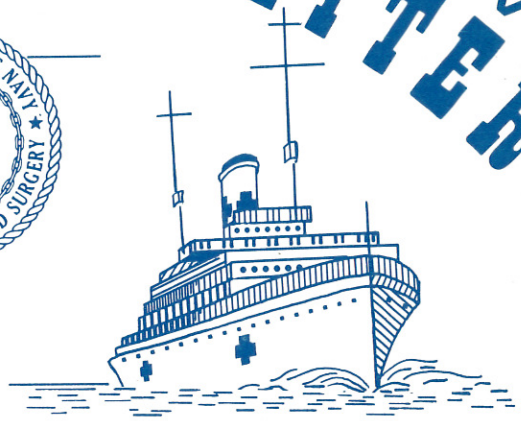

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Policy

The U.S. Navy Medical News Letter is basically an official Medical Department publication inviting the attention of officers of the Medical Department of the Regular Navy and Naval Reserve to timely up-to-date items of official and professional interest relative to medicine, dentistry, and allied sciences. The amount of information used is only that necessary to inform adequately officers of the Medical Department of the existence and source of such information. The items used are neither intended to be, nor are they, susceptible to use by any officer as a substitute for any item or article, in its original form. All readers of the News Letter are urged to obtain the original of those items of particular interest to the individual.

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The issuance of this publication approved in accordance with NAVEXOS P–35.

A FORTUITOUSLY CONTROLLED STUDY OF STEROID THERAPY IN ACUTE VIRAL HEPATITIS*

II. FOLLOW-UP EXAMINATION OF 202 PATIENTS

Rén Stutz, MD, André L. Blum, MD,† Urs Peter Haemmerli, MD, Paul Schmid, PhD and Martin Schmid, MD, Zurich, Switzerland. *Amer J Med 47(1):93–100, July 1969.*

Follow-up physical examination and liver function tests were performed in 202 patients one to seven years after their recovery from acute viral hepatitis. They had been treated in either of two hospitals in which treatment was comparable except that steroids were used frequently (89.5 percent) at one (sixty-three patients) and less often (16.5 percent) at the other (139 patients).

Chronic hepatitis was found in six of eleven elderly women whose acute hepatitis had been mild and protracted. In other patients chronic hepatitis developed more rarely (1 percent). The incidence of chronic hepatitis detected was about the same regardless of the interval between the second examination and recovery from the acute disease. The overall incidence of chronic liver disease was 6 percent in the "steroid hospital" and 7 percent in the "control hospital," but the latter figure was only 3 percent when cases of fatty liver and nonspecific parenchymal changes were excluded. Subjective symptoms were also distributed equally between the hospitals, 62 and 60 percent at the steroid and control hospitals, respectively, and bore little relation to the incidence of chronic liver disease.

The results of the first part of this study suggest that complications of steroid therapy more than offset the short-term advantages when used in viral hepatitis. The question remains whether steroids prevent late sequelae. Both an increased and decreased incidence of chronic liver disease has been reported to follow steroid treatment. A third group is of the opinion that acute hepatitis rarely if ever progresses to chronic liver disease, irrespective of therapy. In our study we attempted to determine whether there was any difference in the incidence of hepatitis sequelae between patients with hepatitis who were treated with steroids and those who were not.

A unique opportunity to evaluate the effects of steroid therapy arose in Zurich because all of the city's patients with hepatitis were admitted to either of two hospitals in which treatment was comparable except that steroids were used routinely at one and seldom at the other.

Methods

A detailed description of the methods used for evaluation of treatment effects has been given in the first part of this study. The designation of the two study hospitals utilized in the first part has been continued. "Steroid hospital" is the City Hospital in which 89.5 percent of the patients received an uninterrupted course of 150 mg or more of prednisone during the first week of steroid treatment, and "control hospital" is the University Hospital in which only 16.5 percent were so treated. Supportive treatment was otherwise comparable.

Patients Followed. We anticipated that follow-up would have to be quite complete in order to draw conclusions about the relative incidence of sequelae that might be encountered only rarely in either group. Therefore we selected only Swiss patients because we thought we could reach most of them; we were successful in contacting all of the 102 patients from the steroid hospital and 236 of 240 at the control hospital. The patients had been hospitalized for their acute hepatitis at some time during the seven year period, 1958 through 1964 inclusive. In January 1966 a questionnaire was sent to all these patients. Of the 338 responses, 310 were from the patients themselves, seventeen from relatives who stated that the patient was living and asymptomatic and eleven from relatives who stated that the patient had died. None of the deaths was attributed to hepatitis or its sequelae and this was confirmed by autopsy in nine of eleven instances.

The questionnaire inquired about many vague symptoms, the more specific of which were involun-

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tary weight loss of more than 3 kg in the most recent follow-up year, acquired intolerance of fatty or specific foods, chronic fatigue and abdominal discomfort. Patients were considered symptomatic if they replied affirmatively to any one of these (Table II). Severe symptoms were defined as those requiring continuous visits to a physician and preventing the patient from performing the daily activities possible before onset of acute hepatitis. All patients were invited to return to our clinics for physical examination regardless of whether their response had been graded as symptomatically positive or negative.

TABLE II. *Frequency of Clinical Relapses and Prolonged Convalescences: Steroid Hospital Versus Control Hospital*

Convalescence	Total		Steroid Hospital				Control Hospital			
					Total				Total	
	No.	%	Steroids	No Steroids	No.	%	Steroids	No Steroids	No.	%
Patients examined	202	100	56	7	63	100	22	117	139	100
Clinical relapses	8	4	5	0	5	8	3	0	3	2
Duration of convalescence > 10 weeks	30	15	9	1	10	16	8	12	20	14

In February and March 1966, 63 percent of the original patients from the steroid hospital and 61 percent of those from the control hospital returned to our clinic for a checkup. The groups were well matched with regard to sex (62 percent in each group were female), age (steroid hospital = 43.0± 1.9 years versus control hospital = 42.3± 1.0 years, mean ± standard error), and prior duration of jaundice and hospitalization.

A complete physical examination was carried out; special attention was given to the abdominal examination and search for spider angiomas or jaundice. Liver function tests were performed on each patient as follows (values in parentheses considered to be abnormal): serum bilirubin (more than 1.3 mg per ml), SGPT (more than 45 Wroblewski units), SGOT (more than 45 Wroblewski units), alkaline phosphatase (more than 6 Bodansky units), bromsulfalein retention (more than 8 percent after forty-five minutes), serum electrophoresis gamma globulins (more than 22 percent of total proteins).

Abnormal laboratory results were rechecked after two to three weeks. If any one of the values was abnormal twice, a liver biopsy was performed. In patients with hepatomegaly (defined as liver edge extending more than 7 cm below the right costal margin at the mid-clavicular line in deep inspiration) the liver was biopsied regardless of the biochemical data. Subjective symptoms were not considered an indication for biopsy. Biopsies were interpreted without knowledge of prior treatment or clinical course.

Results

Only the 202 patients who were reexamined at our hospitals will be considered for the critical evaluation of long-term effects of treatment. Table II shows that these 202 patients were characteristic of the original study population described in the first part of this report. Accordingly, clinical relapses were more common at the steroid hospital, but prolonged convalescence from the initial attack was seen equally often at the two hospitals.

Chronic Liver Disease—Results of Biopsies. Suction needle biopsy was performed in each of the seventeen patients who had at least one abnormal liver function test on two occasions two weeks apart. We had also planned to perform a biopsy in anyone with hepatomegaly but all four of these patients were already included by the foregoing criteria. The predominant biochemical dysfunction was an abnormal bromsulfalein retention or SGOT level (Table III). The serum total bilirubin level was elevated in only one patient with chronic hepatitis (2.0 mg per 100 ml) and in one patient (1.8 mg per 100 ml) with Gilbert's disease and normal histology.

In three patients (including the one with Gilbert's disease) the biopsy specimen was normal and liver function tests repeated after biopsy failed to demonstrate the mild abnormalities encountered on the two occasions before biopsy.

Abnormal histology was found in fourteen patients, eight of whom showed chronic hepatitis including one with transformation to postnecrotic cirrhosis. The findings in these patients are cited in Table III.

TABLE III. *Incidence of Chronic Liver Disease: Steroid Hospital Versus Control Hospital*

Chronic Liver Disease	Total		Steroid Hospital				Control Hospital			
					Total				Total	
	No.	%	Steroids	No Steroids	No.	%	Steroids	No Steroids	No.	%
No. of patients examined	202	100	56	7	63	100	22	117	139	100
Normal liver function tests	185	92	51	7	58	92	19	108	127	91
Abnormal liver function tests	17	8	5	0	5	8	3	9	12	9
Normal liver biopsy	3	1	1	0	1	2	0	2	2	2
Abnormal liver biopsy	14	7	4	0	4	6	3	7	10	7
Chronic hepatitis	7	3	4	0	4	6	0	3	3	2
Chronic hepatitis and postnecrotic cirrhosis	1	1	0	0	0	0	1	0	1	1
Fatty liver	4	2	0	0	0	0	2	2	4	3
Nonspecific	2	1	0	0	0	0	0	2	2	1

Bromsulfalein retention was abnormal in seven of these eight patients, gamma globulin levels were abnormal in six and transaminase levels in four. In none was there evidence of previous serum hepatitis. Six of these eight patients were women over sixty years of age. All six had a characteristic pattern during the acute stage of their illness. Their liver function had been abnormal longer than average, but the serum bilirubin levels had never risen above 11.2 mg per 100 ml or the serum transaminase levels above 650 W.E. However, no clinical or biochemical signs of chronic liver disease had been present at that time. The liver biopsy which had been performed in three patients disclosed the typical picture of acute viral hepatitis. In one female the progression to chronic liver disease could be followed. Chronic hepatitis was evident on biopsy three months after the acute stage and cirrhosis developed within eight months. In two of the six women chronic rheumatoid arthritis had preceded the acute hepatitis. Of the 202 patients examined eleven were women over sixty years old with a mild and protracted course of acute hepatitis. Six of them (55 percent) had chronic hepatitis on follow-up. Of the 191 other patients only two (1 percent) had chronic hepatitis on follow-up. This difference is statistically significant ($p < 0.01$).

Fatty liver was found in four patients. All had abnormal bromsulfalein retention, in one transaminase levels were elevated, but none had elevated gamma globulin levels. None was an alcoholic. Their acute viral hepatitis had been severe and superimposed on serious pre-existing diseases such as breast cancer and Buerger's disease.

Both patients with nonspecific parenchymal liver damage had hepatomegaly and an erythrocyte sedimentation rate over 30 mm per hour. One was a diabetic and had elevated transaminase and alkaline phosphatase levels. The other had elevated gamma globulin levels but no other biochemical abnormality.

Correlation of Chronic Liver Disease with Treatment. Steroid Hospital Versus Control Hospital. Chronic hepatitis was found in four of the patients in the steroid hospital and four in the control hospital (6 and 3 percent, respectively, $p > 0.1$). There were two additional patients at the control hospital who were not treated with steroids in whom nonspecific parenchymal damage occurred. Biopsy disclosed four patients who had fatty infiltration of the liver without signs of chronic viral hepatitis. In all four biopsy had been performed at the time of their initial admission and none had shown any fat at that time. All four of these patients were from the control hospital but two of them had been given steroids. None of these four was an alcoholic nor did they or any other patient show any signs of malnutrition originally or at follow-up. Conversely, there was no sign of chronic liver disease in the eight patients known to be alcoholics (ingesting greater than 100 ml of 100 percent ethanol equivalent per day) originally and continuously up to the time of examination.

There was no correlation between the incidence of chronic liver disease and time interval between hepatitis and follow-up. Chronic liver disease was not correlated with duration of convalescence, diet and intercurrent diseases after the acute hepatitis.

Subjective Symptoms. Steroid Hospital Versus Control Hospital. Correlation of subjective symptoms with steroid treatment was investigated by comparing the questionnaire responses of patients who agreed

TABLE V. *Subjective Symptoms Following Hepatitis as Reported on Questionnaire and Prior to Physical Examination**

Data and Subjective Symptoms	Total		Steroid Hospital				Control Hospital			
					Total				Total	
	No.	%	Steroids	No Steroids	No.	%	Steroids	No Steroids	No.	%
No. of patients examined	202	100	56	7	63	100	22	117	139	100
No symptoms	78	39	23	2	25	40	9	44	53	38
Symptoms										
Mild	86	42	25	5	30	47	11	45	56	40
Severe	38	19	8	0	8	13	2	28	30	22
Subjective symptoms										
Abdominal discomfort	87	43	28	3	31	49	12	44	56	40
Intolerance to fat	80	40	16	4	20	32	10	50	60	43
Intolerance to other foods	49	24	14	2	16	25	7	26	33	24
Fatigue	26	13	5	1	6	10	4	16	20	14
Involuntary weight loss	19	9	7	0	7	11	4	8	12	9

*There is no difference between hospitals in the frequency of positive replies to any of the symptoms queried.

to return for examination. The replies of patients originally admitted to the steroid hospital were contrasted with those of patients originally admitted to the control hospital. The tabulation and comparison was carried out prior to examining the patients and the results are summarized in Table V. We had first determined that the frequency and distribution of positive responses on the questionnaire among examined patients was typical of the entire study population surveyed. The only selection factor was that 62 percent of the patients who reported for examination were female which was a slight but significantly greater representation than in the total population queried (57 percent).

The most frequent symptoms were abdominal discomfort (found in 43 percent of the patients examined) and acquired fat intolerance (40 percent). Acquired intolerance of any other specific food was less frequent (24 percent), as was fatigue (13 percent) and involuntary weight loss of more than 3 kg per year (9 percent). The incidence and severity of symptoms was not significantly different among the patients in the two hospitals. Neither could the symptoms be correlated with any of the following: sex, age, serum hepatitis, severe or protracted jaundice, or length of interval between hepatitis and follow-up. On the other hand symptoms were correlated with dietary habits. Thirty-eight percent of the asymptomatic and 65 percent of the symptomatic patients claimed that since the onset of hepatitis they avoided specific foods, usually fatty ones. It was difficult to determine whether the avoidance was of their own choosing or was recommended by their private physician. For whatever reason, the avoidance "diet" was still being followed at the time of examination by 18 percent of the asymptomatic patients, 45 percent of the patients with mild symptoms and 69 percent of those with severe symptoms ($p < 0.001$).

Subjective Symptoms. Relations to Chronic Hepatitis. In order to find out whether the diagnosis of chronic liver disease could be made from the history, the questionnaires were analyzed without knowledge of physical, biochemical and histologic findings. From the history alone, thirty patients were suspected of having chronic liver disease, mainly on the basis of chronic fatigue, weight loss and abdominal discomfort. The incidence of chronic liver disease among these patients was slightly higher than among the remaining 172 (13 and 6 percent, respectively) but the numbers are too small to be significant statistically. However, six of the fourteen patients with histologically proved chronic liver disease were asymptomatic, and in thirteen the diagnosis had not even been suspected before follow-up.

Comments

Because occasional patients with chronic hepatitis showed symptomatic and biochemical improvement following steroid therapy, early investigators speculated that such treatment might also prevent acute hepatitis from becoming chronic. However, the primary evidence that this could be accomplished was based on the impression that steroid treated patients returned to the hospital with chronic hepatitis less often than expected.

The discovery that steroids are associated with a higher rate of relapse and progression of the disease despite an improvement of biochemical values led to a more cautious view. Moreover, in recent studies with attempts at complete follow-up, the incidence of chronic liver disease following steroid treatment has increased. However, many of these patients were malnourished or had had previous episodes of malnutrition. As if the problem were not complicated enough, the progression of acute hepatitis to chronic liver disease has itself become a controversial issue. There is increasing evidence that such a progression happens only rarely.

In our study chronic hepatitis following acute hepatitis was observed in 4 percent of the cases. However, chronic hepatitis was found in six of the eleven women over age sixty in whom the acute hepatitis had been mild and protracted. It was rare in all other classes of patients, being observed in only two of 191 (1 percent), thus confirming similar findings in other surveys. The same trend was found when only patients who had an acute phase biopsy were considered, i.e., six of the eleven elderly women and seventy-two of the 191 remaining patients. Only one of the seventy-two latter patients was found to have chronic hepatitis on second biopsy, whereas it was found in three of the six women, all of whom had only acute changes in the initial biopsy. These results are consistent with the higher than expected frequency of subacute necrosis following acute hepatitis in postmenopausal women as reported by Jerslid and Klatskin.

It is interesting to note that the prevalence of chronic hepatitis was about the same regardless of how many years after the acute disease the follow-up examination was conducted. This was especially true for the elderly women. We wondered whether these observations represented the resultant of two trends, an exponential decline in the number of persistent hepatitis cases, and the appearance of chronic disease from latent infection. Alternatively, the data could be explained by assuming that the tendency toward chronicity was a host-specific phenomenon somewhat independent of the nature of the acute hepatic inflammation. Finally, the chronic pattern could be only coincidental and unrelated to the acute disease. We could follow the progression of acute to chronic liver disease closely in only one patient, a sixty-three year old woman. Liver biopsy disclosed signs of chronic hepatitis at three months and postnecrotic cirrhosis within eight months.

No evidence was found that steroid treatment can prevent the development of chronic liver disease. There was in fact a tendency toward a higher incidence of chronic hepatitis in the steroid hospital than in the control hospital, although the difference is not significant statistically (6 and 3 percent, respectively, $p > 0.1$). The over-all incidence of chronic liver disease, including fatty liver and nonspecific parenchymal damage, was similar in both the steroid and control hospitals (6 and 7 percent, respectively). There was no evidence that fatty liver or nonspecific liver damage was related to acute viral hepatitis.

The incidence of subjective symptoms following acute hepatitis was surprisingly high when compared with the low rate of chronic liver disease in the same patients. Sixty-one percent complained of symptoms attributed to acute hepatitis, principally abdominal discomfort associated with nausea, bloating, heartburn and fat intolerance. These symptoms are known to be common in any hospital population and also among the general population. It appears that the incidence with which symptoms are observed depends largely upon the persistence of the interviewer. In our study these symptoms did not correlate with treatment of the acute hepatitis. Furthermore, they were a poor indicator of the severity of hepatitis, the interval between hepatitis and follow-up, and the presence of chronic liver disease. On the other hand, subjective symptoms were frequent among patients who were on a diet. Symptoms commonly occurred after eating foods which had been specifically restricted by the family doctor. Dietary restrictions may transform the patients into diet cripples who suffer from subjective symptoms whenever they try to deviate from the prescribed regimen. Therefore, we believe that dietary restrictions should be recommended only in special circumstances.

In Figure 2 (not shown) the results of the two parts (I and II) of this study are summarized. On the average one would do well to "break even" when giving steroids. The occurrence of chronic liver disease following acute viral hepatitis is rare, subjective symptoms are frequent and neither can be prevented with steroid treatment during the acute phase of the illness.

(Tables I, IV, figures and references may be seen in the original article.)

RIGHT AORTIC ARCH AND COARCTATION OF THE AORTA*

*LCDR Marvin Grossman, MC USN** and CAPT William J. Jacoby, Jr., MC USN,*
Dis Chest 56(2):158–160, Aug. 1969.

A young girl with a right aortic arch and aberrant left subclavian artery coexistent with coarctation of the aorta is described. An associated interesting finding was retrograde flow in the left vertebral artery producing a "subclavian steal."

The incidence of right aortic arch in both radiographic and necropsy series is approximately 0.1 percent. Stewart et al have classified this anomaly into three major types: (1) with mirror image branching (origin of vessels is a mirror image of normal left aortic arch), (2) with an aberrant left subclavian artery, and (3) with the latter vessel no longer connected to the arch. Each of these types may have either a right or left ductus arteriosus or on rare occasions, bilateral ducti.

Right aortic arch may occur as an isolated abnormality or in association with congenital heart disease. Although the type with an aberrant left subclavian artery is probably most prevalent, the lack of concomitant cardiac malformations makes it less likely to be included in large series. Contrarily, right arch with mirror image branching is associated with cyanotic heart disease in almost all instances. Tetralogy of Fallot, truncus arteriosus and transposition of the great arteries are found most frequently.

Isolated instances of right sided arch and coarctation of the aorta have been described in the literature. In both cases, the coarctation was coexistent with the Type II or aberrant left subclavian variety.

Case Report

This 14-year-old girl was referred to the Naval Hospital, Great Lakes, Illinois for evaluation of a heart murmur detected on a routine school physical examination. There was no previous history of cardiovascular complaint.

The pertinent physical findings included the following blood pressures in mm Hg: right arm 140/90;

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The opinions expressed herein are those of the authors and cannot be construed as reflecting the views of the Navy Department or of the Naval Service at large.

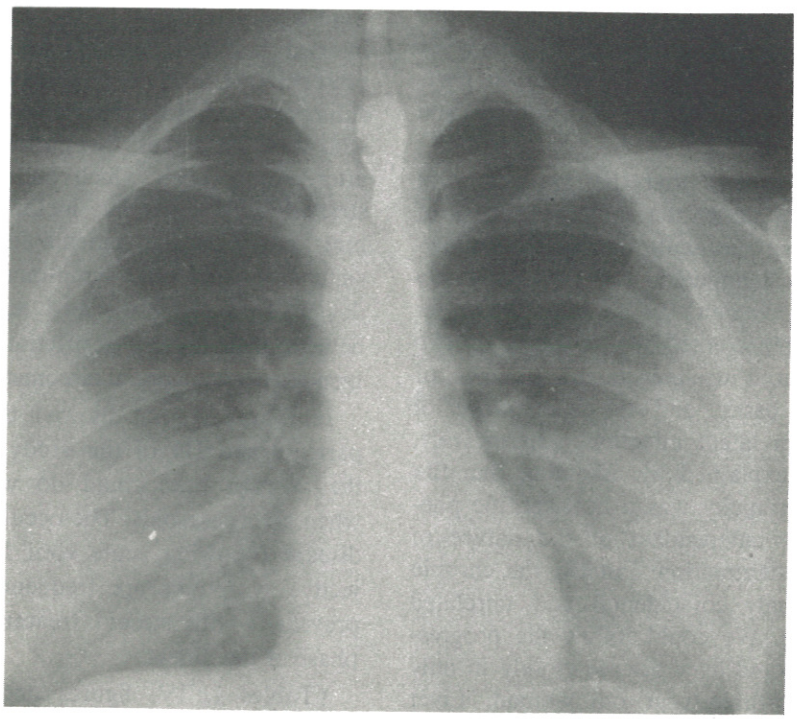

Figure 1. Thoracic roentgenogram shows displacement of barium filled esophagus by right aortic arch. Also note vascular groove produced by abberant left subclavian artery.

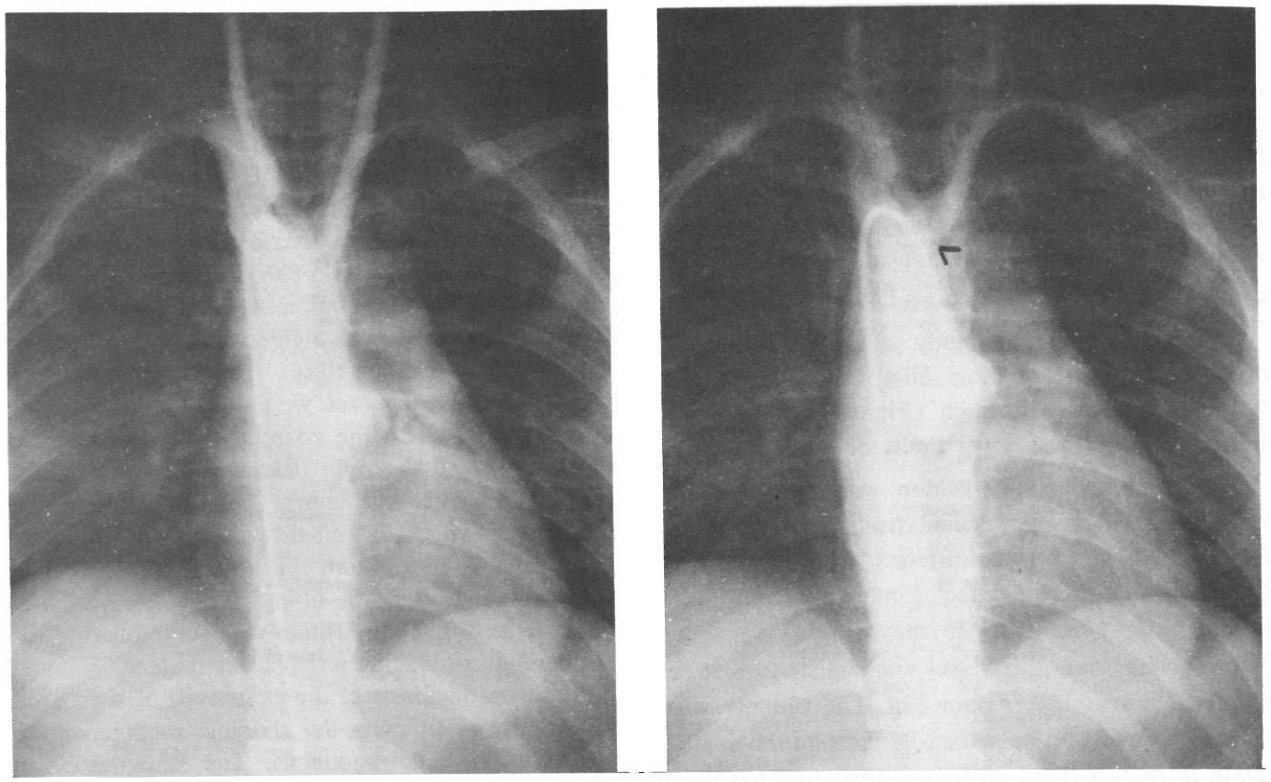

Figure 2A (left). Serial aortic angiogram delineating the left common carotid, right common carotid and dilated right subclavian artery, all of which arise proximal to the coarcted segment. *2B (right).* X-ray of film taken 0.5 seconds later. Lines show origin of left subclavian artery.

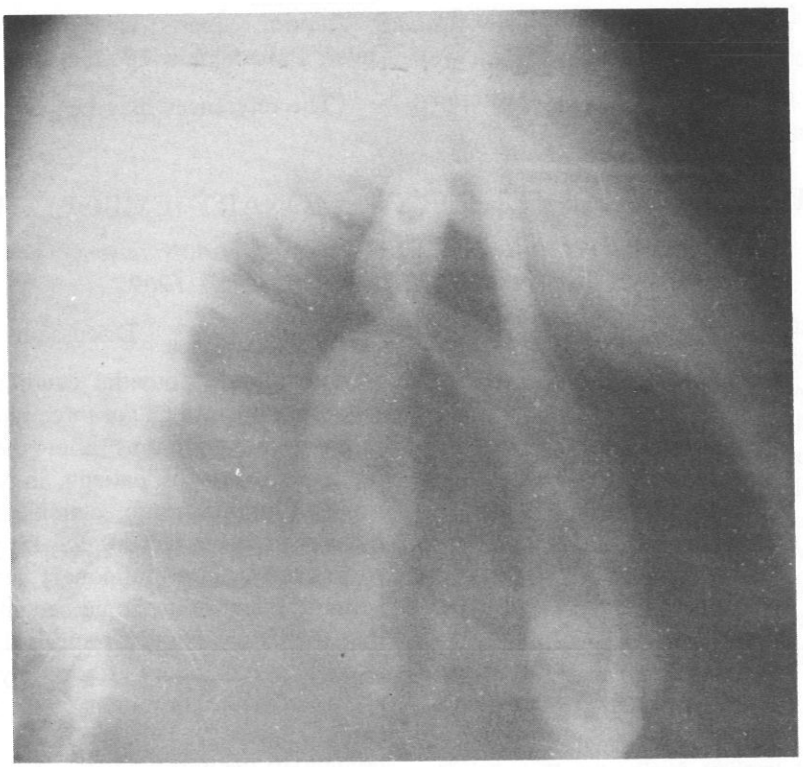

Figure 3. Lateral view of coarctation with catheter in left ventricle.

left arm 90/60; both lower extremities, 110/100. A grade III/VI harsh systolic murmur was heard over the right 2nd intercostal space and radiated to the neck vessels. The murmur was equally audible in the interscapular area. The peripheral pulses (graded on a scale of 0–4) were as follows: right brachial +3; left brachial +1; both femorals and distal lower extremity pulses +1.

The roentgenographic study of the heart showed displacement of the barium filled esophagus to the left by the right aortic arch (Fig 1). The cardiac silhouette was at the upper limits of normal.

A right heart catheterization was normal. The left ventricle was approached from both the right femoral and right brachial arteries. Upon ascending the thoracic aorta, the catheter entered the left subclavian artery just distal to the coarcted segment. The pressure wihin this vessel was similar to that of the distal aorta: 100/70 mm Hg. The catheter was then withdrawn and passed easily through the coarctation to the proximal ascending aorta and left ventricle. The pressures in the latter two chambers were 145/80 and 150/8 mm Hg respectively, resulting in a systolic gradient across the coarcted segment of 45 mm Hg. Angiographic studies were obtained before completion of the procedure (Fig 2 and 3).

Surgical correction of the coarctation was not attempted for fear of compromising the posterior circulation to the brain.

Discussion

The patient differs from the previously recorded cases of right aortic arch with coarctation in her unique anatomic variation. The branching of the major vessels from the arch is the mirror image of the more common left aortic arch with an aberrant right subclavian artery. The first vessel to arise proximal to the coarctation was the left common carotid, followed by the right common carotid, and the right subclavian artery. The latter vessel, as well as the right vertebral, were dilated at their points of origin. Distal to the coarctation at the junction of the arch and the right descending aorta, the left subclavian arose and course posterior behind the esophagus. There was no diverticulum noted at the origin of this vessel from the aorta. An interesting finding was the lack of filling of the left vertebral artery in an antegrade fashion with subsequent retrograde filling producing a "subclavian steal."

Accurate diagnosis of the exact type of right arch may be made utilizing the thoracic roentgenogram of the barium filled esophagus. The configuration in this case of a shallow retroesophageal groove which runs upward and to the left at an approximate 70° angle with the horizontal, conforms to the extremely rare right aortic arch with aberrant left subclavian artery and right ductus arteriosus.

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(The references may be seen in the original article.)

SELECTION OF PATIENTS FOR PULMONARY EMBOLECTOMY*

*Herbert L. Fred, M.D., F.C.C.P.** and Ethan A. Natelson, M.D.†,* *Dis Chest 56(2):139–142, August 1969.*

Despite a renewed and expanding interest in pulmonary embolectomy, criteria for selection of patients remain controversial. Indeed, the wide divergence of opinion is difficult to reconcile. One author states, "All but minor emboli should now be treated by pulmonary embolectomy." Others believe, "The successful completion of emergency angiography virtually precludes the need for embolectomy." We examine here commonly cited indications for pulmonary embolectomy and demonstrate the need for flexibility in their application.

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Discussion

We separate potential candidates for pulmonary embolectomy into one of three types, depending upon duration of symptoms after the embolic episode. Type I consists of patients in whom death ensues rapidly. In this group, classification often must be retrospective. About 50 to 75 percent of patients with massive, fatal pulmonary thromboembolism die within the first hour. Some become agonal so quickly that their only hope for survival would seem to be immediate pulmonary arteriotomy performed without angiographic confirmation of the diagnosis or use of cardiopulmonary bypass. Four of 42 patients so treated left the hospital without cerebral damage.

Type II patients survive the immediate throm-

boembolic insult, thereby providing the physician with time to establish a precise diagnosis and to institute a planned therapeutic approach. The bulk of medical literature on pulmonary embolectomy concerns patients of this type.

Patients in the Type III category have chronic obstruction of their major pulmonary arteries. Only a few of them have undergone embolectomy. Indications for the procedure in this group are especially difficult to define. Nevertheless, the operation has alleviated the chief manifestation of dyspnea on exertion, returned the angiographic appearance of the pulmonary arterial tree to normal, and reduced the degree of pulmonary arterial hypertension.

Our comments hereafter pertain to indications for pulmonary embolectomy in the Type II patient. Currently, angiography is indispensable for establishing the presence of surgically accessible pulmonary thromboemboli in this group. Clinical findings alone are deceptive and have led to unnecessary pulmonary arteriotomy with disastrous consequences.

The angiographic demonstration of massive pulmonary thromboembolism (occlusion of greater than 50 percent of the pulmonary arterial tree) is not necessarily an indication for embolectomy. Angiograms performed serially have demonstrated spontaneous resolution of large pulmonary thromboemboli. Moreover, we have observed angiographic evidence of massive pulmonary thromboembolism in acutely ill patients who have survived without embolectomy.

Systemic arterial hypotension unresponsive to vasopressor therapy is the most frequently cited and probably the most reliable indication for pulmonary embolectomy. But how long should one administer vasopressors before deciding upon such operative management? Some investigators suggest that 20 to 30 minutes constitute an adequate trial; however, patients with systemic arterial pressures recorded as low as 50/0 mm Hg have received vasopressors for at least an hour and have survived without embolectomy.

In some patients pulmonary embolectomy should be done ideally before the onset of systemic arterial hypotension. Support for this contention comes from observations made at Ben Taub General Hospital on nine critically ill patients treated by embolectomy for acute, massive, pulmonary thromboembolism (Table 1). Of the four patients (Cases 2, 7, 8, 9) normotensive at the time of operation, three survived the procedure and are still living, six months to five years later. By contrast, only one (Case 1) of the five patients hypotensive at the time of embolectomy survived the operation. Baker and Wagner have had a similar experience. In their nine cases of embolectomy for acute, massive, pulmonary thromboembolism, the three surviving patients were normotensive, whereas the six who died were hypotensive before induction of anesthesia. We cannot dismiss the possibility that the survivors among their patients and ours might have lived without embolectomy. Yet in our patients, the combined clinical, hemodynamic, and angiographic findings were sufficient to convince us that death was imminent.

TABLE I. *Hemodynamic and Angiographic Data in Nine Cases of Pulmonary Embolectomy*

Case	Age (yr), Sex	Blood Pressure—mmHg. Systemic	Right Ventricle	Pulmonary Artery (mean)	Estimated % Pulmonary Vasculature Occluded
*1	54M	90/60			no angiogram
**2	64F	160/80	80/20	90/26(44)	65
3	88M	100/60	48/12	51/19(27)	85
4	47F	30/0			90
5	82M	70/0			90
6	46F	85/50	75/16	72/25(34)	65
7	50F	130/70	60/15	65/29(39)	60
**8	49F	145/90	70/6	65/17(36)	60
**9	63F	115/75	39/14		60

*Died 6 weeks postoperatively. Cause of death was not evident at autopsy.
**Alive.

Another point deserves emphasis. Two of our normotensive patients (Cases 7 and 8) showed significant clinical and angiographic worsening despite optimal heparin therapy for five and six days, respectively. One of these (Case 8) also underwent a second, preoperative hemodynamic study which demonstrated further rise in the already elevated right ventricular and pulmonary arterial pressures. We, like others, believe that such serial measurements, though not always possible or necessary, provide excellent evidence on which to base decisions for or against pulmonary embolectomy.

Several investigators require the demonstration of either elevated right ventricular end-diastolic pressure or increased "mean right ventricular pressure" before recommending embolectomy. A number of patients, however, with elevated right ventricular pressure have survived without embolectomy. *Conversely, the need for embolectomy may exist even when pressures in the pulmonary artery and right side of the heart are normal.* We have observed two patients with acute, massive, fatal pulmonary throm-

boembolism in whom right ventricular and pulmonary arterial pressures were normal, but the brachial arterial pressure was 80/50 mm Hg. In retrospect, pulmonary embolectomy might have helped these patients.

Some authors stress severe pulmonary arterial hypertension, with or without elevated right ventricular end-diastolic pressure, as an indication for pulmonary embolectomy. Others emphasize a mean pulmonary arterial pressure greater than 30 percent of mean systemic arterial pressure as a basis for such surgical intervention. Nevertheless, patients with massive pulmonary thromboembolism, severe pulmonary arterial hypertension, and/or systemic arterial hypotension have recovered without embolectomy.

The advent of fibrinolytic activators ultimately may affect the selection of patients for pulmonary embolectomy. At present, however, we believe that available information is insufficient to permit firm conclusions about indications for these agents in therapy for pulmonary thromboembolic disease. These drugs, with special reference to urokinase, undoubtedly facilitate dissolution of recently formed pulmonary thromboemboli, but their administration has not brought about significant change in the mortality rate. Furthermore, urokinase is not widely available and its use carries the ever present risk of hemorrhagic diathesis.

Summary

1. From a review of medical writings on pulmonary embolectomy and analysis of our own experience with such cases, we caution against strict adherence to a single criterion or set of criteria for the operation.

2. Systemic arterial hypotension unresponsive to vasopressor therapy is the most frequently cited and probably the most reliable indication for pulmonary embolectomy. In some patients, however, the procedure should be done ideally before the onset of systemic arterial hypotension, particularly when hemodynamic and angiographic studies demonstrate progressive worsening despite optimal anticoagulant therapy.

3. Embolectomy may be necessary even when pressures in the pulmonary artery and right side of the heart are normal.

4. Angiography is indispensable for establishing the presence of surgically accessible pulmonary thromboemboli and should precede embolectomy in all but patients who rapidly become agonal.

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(The references may be seen in the original article.)

SKIN, HEREDITY, AND CANCER

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Hereditary factors in cancer in humans recently have been receiving increasing attention. In spite of significant advances in understanding the genetic factors important in the etiology of many cancers and cancer-predisposing diseases, surprisingly little interest has been given to application of this knowledge to cancer control. Because of ease in observation of lesions and in biopsy confirmation of disease, the cutaneous system lends itself well to investigation and application of genetic information for cancer control. Many hereditary cancers involve the skin either directly, or indirectly, through the presence of distinguishing dermal lesions which may be precursors or concomitant with cancers of other anatomic sites. All presently known dermatologic conditions associated with cancer which have a definite or presumptive hereditary etiology are reviewed and arranged according to specific mode of inheritance. Such information could be beneficial to the physician. All relatives of his patients with a known hereditary cancer or hereditary disorder predisposing to cancer or associated with cancer should be urged to have an early medical examination.

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A possible hereditary etiology of human cancer has been considered intermittently for at least the past 2 centuries. For example, in 1802, a questionnaire sent to all physicians in England contained the question, "Is cancer hereditary?" No firm results apparently emerged from this survey. Early cancer genetic studies involved specific anatomic sites such as skin, breast, stomach, and colon. However, these investigations were glaringly unsystematic and invariably lacked adequate controls. No wonder, therefore, that the hereditary issue in cancer etiology has been steeped with controversy, even to the present time.

The relative importance of cultural and environmental factors in cancer-genetic etiologic hypotheses has been justly a constant source of argument. For example, Bashord, in 1908, demanded that environmental and cultural factors be weighed heavily when interpreting the extraordinarily high frequency of skin cancer, "Kangri cancer," in the Kashmiris of India. He found that the Kashmiris wore a charcoal oven against their abdominal wall for warmth and that the sites of contact with this oven, namely the abdomen and medial thighs, were those where skin cancer most often occurred. We now know of many additional environmental causes of skin cancer, including solar radiation, x-ray, radium, frost bite, exposure to tar, soot, lubricating oil and other hydrocarbons, creosote, paraffin, and arsenicals.

Progress has been rapid in medical genetics since the rediscovery of Mendel's law in 1900. This has led to the creation of numerous genetic subspecialties including molecular genetics, biochemical genetics, immunogenetics, human cytogenics, and highly sophisticated statistical approaches facilitated by the newly emerging computer sciences. However, in spite of these prodigious advances, relatively little progress has been made in human cancer genetics.

Undoubtedly, the complexity of cancer in humans, with mandatory histologic verification of the disease, coupled with man's biological, psychological, and sociological peculiarities, collectively cause him to be a poor subject for cancer genetic studies. For example, problems which pose particular difficulty for genetic analysis include the inability to control matings, long generation intervals, difficulty in validating medical and genealogical data from past generations, and excessive population migration and mobility. Finally, attitudes and feelings about cancer, most prominent of which are fatalism and fear, have been obstacles to early cancer detection. In light of the aforementioned problems, it would seem that of all organs and systems, the skin would be unusually well-suited for cancer genetic studies. It is obvious that malignant neoplasms of the skin often have distinctive identifying characteristics; in addition, several syndromes predisposing to cancer of other anatomic sites have highly characteristic dermatologic signs. Finally, skin lesions are usually apparent to the naked eye. Thus, much clinical information which the physician obtains by observing the skin will also often be similarly apparent to the patient and his relatives. Recording and documentation of skin lesions, therefore, could prove of inestimable value in comprehending the role of genetic factors in certain forms of human cancer.

Several hereditary dermatologic disorders associated with cancer as well as specific dermatologic cancers have been described fully in the literature. Surprisingly, however, this subject has never been completely reviewed and presented in a systematic manner. Therefore, the purpose of this report is to present a review of existing evidence for hereditary dermatologic disorders associated with cancer, as well as specific hereditary dermatologic cancers.

Autosomal Dominant Conditions

Neurofibromatosis: Neurofibromatosis is inherited as an autosomal dominant. Affected individuals show an increased risk (5 to 15 percent) for the development of unicentric or multicentric sarcomatous neurofibromas involving the brain, spinal cord, and peripheral nerves. This risk is present no matter how minimal are the stigmata of neurofibromatosis. For example, one patient showed only a few café au lait spots while some of his relatives showed more severe cutaneous manifestations of this disease. However, this patient presented with an inoperable intracranial neurofibrosarcoma. On the other hand, a second unrelated patient, showed disseminated cutaneous involvement by neurofibromatosis. She died at age 64 from heart disease and never developed cancer. There is no way of detecting which patients with neurofibromatosis will develop cancer. Therefore, no matter how minimal the phenotypic manifestations may be in an affected individual, he should be considered at risk for the development of cancer.

Other associated neoplasms which may occur in patients with neurofibromatosis include pheochromocytoma and acoustic neuroma (often bilateral).

Gardner's syndrome: Gardner's syndrome is inherited as an autosomal dominant. Epidermal cysts of the skin are the most common manifestations. Patients freqently manifest a predisposition to osteomas, particularly of the maxilla, mandible, and sphenoid sinuses; lipomas of the skin occur. More

ominously, patients inherit a predisposition for polyposis coli. The incidence of malignant alteration of adenomatous colonic polyps in 118 cases of Gardner's syndrome found in the literature was 45 percent. However, the true incidence of colonic malignant neoplasia with increasing age may approach 100 percent, the same as that given for familial polyposis coli. Other types of cancers associated with this disease are liposarcomas, myxosarcomas, and thyroid cancer. However, due to an insufficient number of reports, their true incidence in Gardner's syndrome cannot be calculated at this time.

A family was recently reported wherein a father and one of his children died respectively from pulmonary osteosarcoma and retroperitoneal liposarcoma; a second child died from carcinoma of the colon with polyposis coli, while a third child had both reticulum cell sarcoma and polyposis coli. It was postulated that the disease in this kindred might possibly have represented a variant of Gardner's syndrome coexisting with tumors of mesenchymal origin.

Multiple nevoid basal cell carcinoma syndrome: Multiple nevoid basal cell carcinoma syndrome is inherited as an autosomal dominant. It is characterized by one or more congenital anomalies. An important cutaneous anomaly consists of multiple basal cell carcinomas which may occur on any skin area with an early age at onset (mean age 15). Other cutaneous anomalies include multiple epithelium-lined cysts, lipomas, and hypokeratinized areas of the palms, "palmar pits," and soles. Dental and osseous anomalies include defective dentition and jaw cysts which are microscopically similar to dentigerous or primordial cysts, bifid ribs, spina bifida occulta, scoliosis, cervical and thoracic vertebral fusion, and brachymetacarpalism; neurologic anomalies include medulloblastomas, dural calcification, bridging of the sella tursica, agenesis of corpus callosum, and mental retardation; ophthalmologic anomalies include hypertelorism, dystopia canthorum, congenital blindness and strabismus; a possible endocrinologic aberration is hyporesponsiveness to parathormone. Finally, women have increased incidences of ovarian fibromas.

Cutaneous malignant melanoma: Cutaneous malignant melanoma may have an hereditary etiology in some families. The hereditary variety of this disease accounts for about 3 percent of all cases encountered, thus suggesting that extra genetic factors are of greater importance in the majority of cases. The hereditary variety has an early age at onset. Calculations by Anderson et al. show that of the 97 reported cases of familial melanoma, the average age at onset was 40.7 $\pm$ 1.7. This was significantly lower ($P<0.01$) than the average of 49.0 $\pm$ 0.5 from a large consecutive series of melanoma patients. It also has an increased occurrence of multiple primary melanomas and behaves as an autosomal dominant. Figure 1 (not shown) illustrates a family in which this disease was present through 3 generations. Therefore, when the physician encounters a patient with the hereditary form of melanoma, he can anticipate the possibility that a number of the patient's relatives will also be affected and, through early detection, he might provide significant improvement in prognosis.

Giant pigmented nevi (bathing trunk nevi): Giant pigmented nevi may involve an extensive cutaneous surface and, in some cases, may involve an entire segment of the body. Their manifestations are variable and range from flat angiomatous, verrucous, and hairy nevi. These nevi may be present at birth, and previously were thought to be benign. However, this reasoning is incorrect since many of the true melanomas of children arise from these lesions.

The role of heredity in this disorder is not clear. However, the occurrence of the lesions at birth and their possible association with neurocutaneous melanosis, and the proven familial occurrence of cutaneous melanomas in at least 3 percent of melanoma patients, suggest the possibility of a genetic etiology.

Tylosis and esophageal cancer (keratosis palmaris et plantaris): Tylosis is a general term denoting hyperkeratosis of a body surface; in keratosis palmaris et plantaris, hyperkeratosis occurs on the palms and soles. This condition has been described in numerous families and was found to be inherited as an autosomal dominant, completely benign. However, an association with esophageal cancer was found in 3 families showing this pattern of tylosis. A life table was constructed for 2 of these families in which it was predicted that 95 percent of relatives with tylosis would develop esophageal cancer by age 65. Autosomal dominant inheritance was observed in these families.

In families showing tylosis and esophageal cancer, individuals manifesting the cancer "marker" (tylosis) should be considered to be at extraordinary risk for development of esophageal cancer and should undergo frequent diagnostic esophageal studies, particularly after the age of 40.

Carcinoma of the esophagus, in the absence of tylosis, does not have a hereditary basis.

Tuberous sclerosis (Bourneville's disease, Pringle's disease): Tuberous sclerosis is inherited as an

autosomal dominant with a wide range of expressivity of the gene. A triad of adenoma sebacea of Pringle involving the face, epilepsy, and mental retardation is the common manifestation of the syndrome. Actually, adenoma sebaceum is a misnomer in that the lesion is more in keeping with a hamartoma in which sebaceous glands are only passively involved.

Tumor-like masses or "hamartomas" may be found in the brain, described as potato-like nodules or "brain stones"; similar lesions are also found in the kidney and heart.

About 1 to 3 percent of patients with tuberous sclerosis develop gliomas; the majority of these tumors are benign giant cell astrocytomas while a lesser number are glioblastoma multiforme, ependymoma, and fibrillary astrocytoma. Since cerebral neoplasms may complicate this disease, any patient showing adenoma sebacea and neurologic signs should be evaluated carefully for the presence of resectable brain lesions.

von Hippel-Lindau's syndrome: The constellation of angiomatosis of the retina and cerebellum, tumors of various organs (hemangiomas and multiple cystadenomas), including cavernous hemangiomas of the face and neck, constitute the von Hippel-Lindau syndrome. This rarely occurring condition is inherited as an autosomal dominant with variable expressivity.

This disorder shows an increased association with hemangioblastoma of the cerebellum, renal cell carcinoma, and pheochromocytoma.

Peutz-Jeghers syndrome (pigmentation-polyposis syndrome): Pigmentation and polyposis are the 2 essential features of the Peutz-Jeghers syndrome. The pigmentation consists of a distinctive pattern of melanin spots distributed on the oral mucosa (most frequently lips and buccal mucosa, less often the gums, hard palate, and only rarely the tongue), distal portions of the fingers, and occasionally the vaginal mucosa. Pigmented areas of the face also may occur concentrated about the mouth, below the nose, and around the eyes. Polyposis may be found at any location in the gastrointestinal tract except the esophagus. The most frequent site is the jejunum (63%) followed by the ileum (55%), colon and rectum (36%), stomach (23%), duodenum (15%), and appendix (3%). Polyps have also been described in the urinary bladder and nasopharynx, though these occurrences have been rare. These hamartomatous polyps consist of layers and projections of mucosa which contain the various types of cells normally present in the affected part and are usually interspersed with bands of smooth muscle.

The most frequent complication of the polyps is intussusception. Malignant transformation in these adenomatous polyps has been an extremely controversial issue with many authorities claiming that it never occurs. However, recent evidence has confirmed metastases in this syndrome presumably from an intestinal polyp. Interestingly, a total of 7 cases of carcinoma of the duodenum has been described. Adenocarcinoma was found in a polyp resected from the transverse colon with invasion of the stalk and bowel wall in a patient, while several other patients with Peutz-Jeghers syndrome have been described with granulosa theca cell tumors.

Peutz-Jeghers syndrome is inherited as an autosomal dominant and at least 300 cases have been described in the literature.

Epidermolysis bullosa dystrophica: Epidermolysis bullosa dystrophica is a rare hereditary disease which may be inherited as an autosomal dominant in some families and as an autosomal recessive in others. The disorder may be first noticed at birth or during infancy. It is characterized by the development of vesicles and bullae, located primarily upon extensor surfaces of joints and other sites which may be exposed to trauma. Mucous membranes may also be involved. The blisters show a tendency to become hemorrhagic followed by bluish atrophic scars. Leukoplakia and carcinoma may occur on mucous membranes; multiple basal and squamous cell carcinomas of the skin have also been described in patients with this disease. Further reporting of associations of cancer with this disease will be necessary before the true relationship can be established.

Kaposi's sarcoma (multiple idiopathic hemorrhagic sarcoma of Kaposi): Kaposi's sarcoma is an obscure disease with distinctive cutaneous manifestations, though other systems including the gastrointestinal tract may be involved.

Cutaneous lesions are frequently found on the extremities, particularly on the legs and feet. However, no area of the skin is immune. Early lesions appear as reddish, purplish, or bluish-brown nodules with rubbery or firm consistency; their distribution may be unilateral but later are often symmetrical. Lesions may appear simultaneously on the hands and feet. The process tends to course along the veins or lymphatics. Purpura, secondary infections, and edema occur frequently. Sarcomatous changes may occur in these lesions. In addition, malignant lymphoma, Hodgkin's disease and leukemia may coexist with Kaposi's sarcoma.

The epidemiology of this disease is still unclear.

There is a significant sex ratio of male to female involvement (about 10 to 1). There appear to be certain geographic, racial, and ethnic predilections with increased occurrences among Northern Italians, Ashkenazi Jews from Russia and Poland, and the Bantus of South Africa. The lesion is about 10 times as frequent among the Bantus as among the whites living in this region and is the most common cancer of the extremities in this group. Multiple familial occurrences of this lesion have been consistent with an autosomal dominant gene showing incomplete penetrance. However, common environmental factors interacting with a susceptible genome must be given careful consideration in any etiologic hypothesis for this disease.

Multiple (generalized) keratoacanthoma: Multiple keratoacanthoma is an exceedingly rare cutaneous lesion which occurs most commonly during adolescence and early adult life. The lesions begin as red macules which progress rapidly to papules and nodules. The mature lesion is usually circular, firm, and pearly in appearance with a rolled and notched rim surrounding an umbilicated crater-like center, which, on gross examination, may mimic squamous cell carcinoma.

An interesting characteristic of the lesion is its propensity for exceedingly rapid growth followed by spontaneous involution. More complete information concerning the nature of this process could provide important clues to carcinogenesis.

While this condition is almost always benign, cases of progression to squamous cell carcinoma have been documented. This condition is inherited as an autosomal dominant.

Autosomal Recessive Conditions

Xeroderma pigmentosum: Xeroderma pigmentosum is a classically inherited (autosomal recessive) precancerous disorder of the skin. While many clinicians believe that this rare disease involves the skin exclusively, in fact, it may present as a chronically progressive multi-system disease. Neurologic manifestations may include speech disturbances, mental deficiency, and convulsive disorders. Ocular manifestations include photophobia, conjunctivitis, keratitis, blepharitis, ectropion, and basal and squamous cell carcinomas. Endocrinologic aberrations include aminoaciduria, prolonged erythrocyte glucose-6-phosphate dehydrogenase (G–6–PD) activity with suggestion of erythrocyte glutathione deficiency, and possible pituitary-adrenal abnormalities. However, the principal target organ is the skin. The skin manifestations are dependent upon age and environment. The pathologic changes are variable but include disturbances of pigmentation and maturation of epidermal cells, leading eventually to the formation of malignant skin tumors. The skin may be dry and scaling with hyperkeratosis. The clinical picture may simulate that of radiodermatitis.

The most frequently occurring skin cancers are basal and squamous cell carcinomas. Malignant melanoma appears to show an increased association with this disease with an estimated involvement in about 3 percent of patients. This disease is of particular etiologic interest since affected patients show an exquisite sensitivity to solar radiation with resultant skin cancer occurring in some patients after only minimal exposure to sunlight.

Normal human skin fibroblasts in tissue culture repair damage to DNA by "repair replication" wherein new bases are inserted into DNA. However, Cleaver has recently demonstrated a failure of repair replication in cells from patients with xeroderma pigmentosum following exposure to irradiation with ultraviolet light. The most important measure for cancer control in this disorder is to protect the individual from the sun's rays.

Werner's syndrome (progeria of the adult): Werner's syndrome is inherited as an autosomal recessive. This disorder begins at about the second decade of life and is characterized by premature senility including premature greying of the hair, baldness, juvenile cataracts, and arteriosclerosis. Systemic problems, possibly of endocrine origin, include an increased incidence of diabetes mellitus, hypogonadism, and short stature.

Premature aging of the skin occurs, attributed mainly to the loss of subcutaneous fat with resultant atrophy, scleroderma-like findings, and circumscribed hyperkeratosis. For a time, Werner's syndrome was considered to be essentially synonymous with Rothmund's syndrome. However, as a result of systematic studies by Thannhauser, these syndromes have been distinguished.

Approximately 10 percent of the patients with Werner's syndrome develop cancer. In a review of 125 cases of Werner's syndrome, Epstein and associates found a striking preponderance of noncarcinomatous neoplasms. Seven of these patients whose ages ranged from 35 to 47 manifested the following tumors: fibrosarcoma, osteogenic sarcoma, sarcoma of nerve sheath origin, melanotic sarcoma, hemangiolipoma with occasional mitosis, spindle cell

sarcoma, and uterine myosarcoma. In addition, 3 patients manifested meningiomas and 4 had carcinomas of the following histologic types: adenocarcinoma of hepatic duct origin, carcinoma of the liver, carcinoma of the breast, and papillary adenocarcinoma of the thyroid. Thus, cancer was observed in 14 of these 125 patients. It was of interest that skin cancer was not found in any of these patients in spite of the frequent occurrence of chronic ulcerations and atrophic changes of the skin.

Ataxia-Telangiectasia (Louis-Bar Syndrome): Four clinical features of ataxia-telangiectasia include: 1. oculocutaneous telangiectasia, 2. cerebellar ataxia, 3. mental and growth retardation, and 4. recurrent sinopulmonary infections. This condition may be recognized first by ataxia of gait when the child is first learning to walk. Cutaneous manifestations are first observed at about the age of 5 years when fine red streaks appear, symmetrically arranged in the temporal and nasal area of the conjunctiva; these may extend later to the butterfly area, the ears, antecubital and popliteal areas, as well as to the dorsum of the hands and feet. These telangiectasias are venous. Patients frequently have lymphopenia and deficiency in developing delayed hypersensitivity (gamma 1-A globulin defect). Detailed necropsy studies of this disease have only become available recently.

Malignant transformation in this disease is primarily of the lymphoreticular variety. Peterson and associates described 6 patients with ataxia-telangiectasia of whom a girl, age 9 years, had lymphosarcoma (necropsy) and a boy, age 5 years, showed reticuloendotheliosis considered to be compatible with histiocytosis-X. In addition, 6 cases of ataxia-telangiectasia with associated malignant tumors have been described in the medical literature. The specific tumors were generalized reticulum cell sarcoma in one, Hodgkin's disease in another; a third had undifferentiated round-cell sarcoma and 3 other children, all siblings, were said to have had malignant tumors of lymphoid tissue. Recently, 2 patients with ataxia-telangiectasia, cancer, and an untoward reaction to radiation therapy have been reported. One patient was a 10-1/2-year-old boy with lymphosarcoma (necropsy) and the second was a 9-year-old boy with Hodgkin's disease (necropsy). Ataxia-telangiectasia is inherited as an autosomal recessive.

Bloom's syndrome (congenital telangiectatic erythema and stunted growth): Bloom's syndrome is an exceedingly rare autosomal recessively inherited disorder which occurs predominantly among individuals of Jewish extraction. Chromosomal abnormalities include a high incidence of chromosome breakage and rearrangements.

The principal cutaneous lesions include congenital telangiectatic erythema of the face, particularly in the "butterfly distribution" with significant sensitivity to sunlight. Indeed, the facial appearance closely resembles that of lupus erythematosus. Erythema may occasionally occur over the dorsum of the hands and forearms. Associated skin anomalies have included café au lait spots, ichthyosis, acanthosis nigricans, hypertrichosis, and keratosis pilaris. In addition, these patients are almost invariably short in stature, slender, with a fine-featured face and a dolichocephalic head.

There is a high predilection for leukemia. For example, 3 of 23 patients with Bloom's syndrome died from leukemia at ages 12, 23, and 25. A fourth patient had lingual carcinoma.

Chediak-Higashi syndrome: The Chediak-Higashi syndrome is inherited as an autosomal recessive. The cardinal feature of the syndrome involves an anomalous granulation in the polymorphonuclear leukocytes in the peripheral blood, inclusion bodies in myeloid series in the bone marrow, and irregular masses resembling Döhle's bodies in the granulocytes. Variable cutaneous manifestations have been described including fair complexion, dilution of hair color, hyperpigmentary response to sunlight, and excessive sweating.

Electron microscopic studies have recently shown a relationship between the pigmentary abnormality in this disorder and gigantism of the melanosomes which is similar to the gigantism of the leukocytic granules. This generalized involvement of lysosomal granules has also been described by Lutzner and associates in Aleutian mink which manifest a disorder quite similar to the Chediak-Higashi syndrome in man.

Findings in other organ systems include retinal albinism associated with photophobia and nystagmus, recurrent infections associated with fevers which may not be related to infection, and, less frequently, hepatosplenomegaly, lymphadenopathy, and neurologic manifestations including mental retardation, convulsions, and neuropathy.

Autopsy studies have shown infiltration in viscera and lymph nodes "interpreted as compatible with a malignant lymphoma." Lymphoma in association with this disease has been described by others. Dent and associates suggest that the lymphoma associated with this disorder may result from an inordinate susceptibility to an infectious agent. Specifically, they have

found abundant virus-like particles in peripheral leukocytes from 2 of their patients. The particles resembled the Bernhard type C virus which has been associated with animal leukemia and lymphoma. They speculate that an increased fragility of lysosomal membranes in certain viral infections might result in an "abnormal release of lysosomal enzymes, which in turn would lead to the development of neoplastic changes within the cell. . . ."

The occurrence of the Chediak-Higashi syndrome, not only in Aleutian mink but also in a herd of albino cattle, has been of interest to geneticists and pathologists, though, to date, lymphoma has not been found in animals with this syndrome.

Albinism: Albinism is characterized by a partial or total absence of pigmentation, most noteworthy in the skin, hair, and eyes ("pink eye"). The melanocytes of the skin are amelanotic. There appear to be several phenotypic varieties of albinism presumably due to different genetic mutations. Thus, some forms of albinism may be of the so-called "pure" type while others may be associated with impairment of vision, hearing, or mental retardation. Inheritance in the majority of cases of albinism is autosomal recessive.

The skin of patients with albinism tends to show premature aging as noted by increased occurrence of actinic cheilitis, telangiectasia, keratoses, and cancer, particularly squamous cell carcinoma. In some respects, the skin parallels that found in xeroderma pigmentosum.

Fanconi's aplastic anemia (congenital pancytopenia): Fanconi's anemia is inherited as an autosomal recessive; in addition, chromosome studies of fibroblasts, lymphocytes, and bone marrow have shown a high incidence of chromosomal structural aberrations including chromatid and isochromatid breaks, exchanges, and endoreduplications.

The cardinal findings in Fanconi's anemia include pancytopenia, bone marrow hypoplasia, and congenital anomalies. The latter may include hypoplasia of the thumbs, absence of the radius, strabismus, microcephaly, microphthalmia, dwarfism, and hypogenitalism. In addition, patients often show a spotty or patchy brown pigmentation of the skin. Indeed, the skin manifestations in some patients may appear similar to those found in patients with dyskeratosis congenita. Some authors consider the conditions identical. Family studies have revealed that relatives in at least one of these families have manifested features of both diseases, suggesting that they are genetically related. The clinical picture is thought to be determined by the extent of mesodermal abnormalities (Fanconi's syndrome) versus ectodermal manifestations (dyskeratosis congenita). Cytogenetic studies of patients with dyskeratosis congenita should add clarification to the classification of these disorders.

Cancer shows an increased occurrence in Fanconi's anemia though existing data is insufficient for calculating meaningful incidence figures. The most frequently occurring malignant neoplasm in this disease is acute leukemia. Interestingly, 2 of the patients described by Bloom and associates died from acute monocytic leukemia 6 months and 6 years respectively after the original diagnosis of pancytopenia. Swift and Hirschhorn, on the other hand, reported a 30-year-old woman with squamous cell carcinoma of the skin of the anus and carcinoma in-situ (Bowen's disease) of the vulva.

Sex-Linked Recessive Conditions

Aldrich syndrome (Wiskott-Aldrich syndrome): The Aldrich syndrome is inherited as a sex-linked recessive. The onset of this disease is in infancy, often earlier than 4 months. The disorder is characterized by generalized chronic eczema, erythroderma, thrombocytopenia, petechiae, purpura, recurrent infections including pyoderma, furuncles, and purulent otitis media with the majority of deaths resulting from overwhelming viral and bacterial infections. A lack of resistance to infection in this disease is thought to be due to a severe immunologic deficiency.

Cancer, particularly of the reticuloendothelial system, has been shown to be increased in the Aldrich syndrome, though true incidence estimates are not yet available. An unusual family has been described by Basel and associates in which 4 brothers, in a sibship of 7, manifested this disease. Two of these brothers died from neoplastic disease; one at age 3 showed findings consistent with myelogenous leukemia (necropsy), while his brother died at age 6 with findings resembling anaplastic reticulum cell sarcoma (necropsy). In their literature review, they found a boy of 20 months and another 2-1/2 years old with malignant reticuloendotheliosis, a boy aged 7 years and 10 months with astrocytoma of the brain, and two 8-year-old boys with malignant lymphoma.

Bruton's sex-linked agammaglobulinemia: Bruton's agammaglobulinemia is inherited as a sex-linked recessive. Affected males begin to show evidence of immunologic deficiency at about 6 months of age as evidenced by multiple recurrent infections such as pneumonia, suppurative otitis media, and

meningitis. Skin manifestations include severe pyodermas, cellulitis, furunculosis, and conjunctivitis. Death often results from fulminant infections.

The pathogenesis of the immunologic deficiency in this disease has been ascribed to a defect in the formation of the immunoglobulin-producing system.

Malignant lymphoma occurs in this disorder with a frequency significantly greater than expected in the general population. Page et al. described 2 children with sex-linked agammaglobulinemia and malignant lymphoma from a population comprising 24 children with congenital agammaglobulinemia. They compared the risk for malignant lymphoma in a comparable pediatric age group from their region and calculated that the risk for this disease in association with sex-linked agammaglobulinemia is highly significant.

Possible Genetic Etiology

Dermatomyositis: Dermatomyositis is a rare disorder characterized by inflammatory and degenerative changes primarily of skin and muscle (leading to atrophy, weakness, and tenderness). Systemic manifestations may occur, including intermittent fever. Skin changes appear and may be heralded by erythema and puffy edematous swellings of the eyelids ("heliotrope bloating"); other cutaneous findings include minute telangiectasia, erythematous patches on the face and extremities (particularly upper) which are often followed by brown pigmentation. These latter findings may mimic those found in lupus erythematosus. Vasomotor disturbances similar to Raynaud's disease may occur. Following exacerbations of dermatomyositis, one may observe superficial epidermal exfoliation, mild pruritus, and occasionally poikiloderma.

There appear to be both a juvenile form (onset before age 20) and an adult form of this disease. Cancer is frequently associated with the adult variety with estimates of associated malignant neoplasms ranging as high as 50 percent. The most commonly occurring types of neoplasms include cancer of the gastrointestinal tract, sarcomas of soft tissues, lymphoma, and multiple myeloma. As in lupus erythematosus, autoimmune factors have been suggested, and it is not certain whether, in certain situations, a malignancy might predispose the patient to dermatomyositis.

The genetics of dermatomyositis are complex and as yet a specific mode of inheritance cannot be determined. Lambie and associates studied a large kindred in which 2 female first cousins manifested this disease. An interesting finding was an increased occurrence of rheumatoid arthritis and certain serum protein abnormalities, thus suggesting a relationship between dermatomyositis and other connective tissue disorders.

Sjögren's syndrome (keratoconjunctivitis sicca): Sjögren's syndrome is a systemic disorder which has been classified by some investigators in the collagen group. The condition has been found to be associated with systemic lupus erythematosus, polymyositis, polyarteritis, scleroderma, and Raynaud's phenomenon. At some time in the course of the disease, the majority of patients will develop rheumatoid arthritis.

Constant features of this disease include keratoconjunctivitis sicca and xerostomia secondary to diminished secretory activity of exocrine glands (lacrimal, salivary, and mucous glands).

The skin is characteristically dry and may show pigmentation including typical café au lait spots. Purpura and telangiectasia of the lips and fingers tips have also been observed.

There appears to be an increased incidence of malignancies of the reticuloendothelial system in this disorder, most frequent of which is malignant lymphoma. In addition, adenocarcinoma was present in the parotid gland of a patient with Sjögren's syndrome.

The inheritance of Sjögren's syndrome is not clear. A strong family history has been described in several instances; reportedly, one family contained 12 affected individuals in 3 generations.

Systemic lupus erythematosus (SLE): SLE is a multi-system disease characterized by intermittent fever, arthritis, arthralgia, arteritis, phlebitis in the central nervous system, renal disease (lupus nephritis), and cutaneous manifestations which include malar erythema distributed in the so-called butterfly area of the face.

Current conceptions of the pathogenesis of this disease strongly impugn autoimmune mechanisms. Genetic factors undoubtedly contribute to many occurrences of SLE with autosomal dominance, the likely mode of inheritance for the hereditary variety.

Evidence for an association between SLE and malignancy has been accumulating rapidly; the most frequent varieties of cancer are thymic tumors and lymphomas. It is possible that, in certain circumstances, cancer might produce a "lupus-like" picture.

Skin Cancer in General

Finally, one must consider the racial and ethnic background as contributory genetic factors in susceptibility to skin cancer. For example, it is well known that individuals who are of fair complexion, with blue eyes and blonde or red hair, and of Nordic extrac-

tion, are significantly more susceptible to skin cancers than those individuals who are heavily pigmented, such as Negroes. As in the case of xeroderma pigmentosum, solar radiation may act in concert with the genotype to produce skin cancer.

Comment

It is apparent from this listing of hereditary skin lesions associated with cancer, as well as specific hereditary skin cancers, that knowledge of the family history coupled with keen observations of the patient's integument, in certain circumstances, could lead to improved cancer control. However, occasionally, cutaneous disorders may occur sporadically in association with cancer and yet mimic the hereditary variety (phenocopy) which may not be associated with cancer. Such an example is found in acanthosis nigricans. The hereditary variety of this disease is inherited as an autosomal dominant and is not associated with cancer. However, acanthosis nigricans may occur sporadically as a result of an internal adenocarcinoma. Thus, in the hereditary form of this lesion, the confirmation of a family history of this skin defect will reassure the physician that he is not necessarily dealing with an underlying adenocarcinoma and therefore save the patient considerable time, expense, anxiety, and discomfort in the search for cancer. On the other hand, should acanthosis nigricans appear sporadically in a patient, it will be strong evidence for the presence of adenocarcinoma. However, it is still possible that acanthosis nigricans in the particular patient actually might be hereditary since it might have resulted from a rarely occurring spontaneous mutation. Other examples of hereditary skin diseases include seborrheic keratosis, in which the association with cancer is unclear. The genetic variety of this disease behaves as an autosomal dominant. Such diseases warrant further investigation in order to discern their possible predisposition to skin cancer.

Finally knowledge of hereditary factors in skin diseases related to cancer will provide the physician with an opportunity to use this information in genetic counseling. No matter how benign a skin lesion might be, if it appears in other members of the family, it arouses many questions and concern. Occasionally, bizarre misconceptions based on folklore and old wives' tales might be harbored by the family, and offered as causes of the occurrence of the particular lesion. Through genetic counseling, the physician can dispel such misconceptions, help to allay anxiety, and place the particular disease in its appropriate biological perspective. The major reward for these efforts will often be reflected by an improvement in cancer control.

(The figure and references may be seen in the original article.)

THE NATURE OF BENIGN STRICTURES IN ULCERATIVE COLITIS*

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Abstract. In a series of 19 examples of benign stricture of the colon in patients with chronic ulcerative colitis insufficient fibrosis was found in the resected specimens to account for the narrowing. Measurements of the various layers of the gut wall were made in both the portions involved and those not involved by stricture, and these were compared with similar measurements in resected specimens from patients with ulcerative colitis without stricture and in normal sigmoid colons. The most striking finding was that in ulcerative colitis the muscularis mucosae is hypertrophied and is in a state of contraction. In the zones of stricture the contraction appears to be so forceful that the inner circular coat of the muscularis mucosae pulls away from the outer longitudinal coat, thereby narrowing the lumen of the gut. The importance of this finding is that such strictures should be reversible.

The pathology of ulcerative colitis as a specific entity has now been established. Its main feature is inflammation of the mucosa, with degeneration of crypt epithelium, often with the formation of crypt abscesses and ulcers.

There is disagreement about the frequency of benign stricture in chronic ulcerative colitis, some authors stressing its rarity and others reporting a high prevalence. The pathology of stricture formation in this disease has received surprisingly little attention,

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and most authors have assumed that stricture formation is due to fibrosis.

The object of this paper is to present evidence that stricture formation in ulcerative colitis is due to hypertrophy and thickening of the muscularis mucosae, and is rarely the result of fibrosis. The importance of this lies in the possibility that the process is reversible.

Material and Methods

In the period 1950–1967, 438 patients with chronic ulcerative colitis were fully studied and documented. Of these, 156 were operated on, and in 19 there were segments in which the diameter of the lumen was reduced by two thirds or more. The specimens were all fixed in 10 percent neutralized formalin in water. The various layers of the gut wall of the stenotic and adjacent nonstenotic segments were measured and compared. Sections of the gut wall were all taken at right angles to the long axis of the colon. The sections were projected onto a screen, and the images measured. Because sections did not always include the taeniae coli, the outer longitudinal muscle coat was not measured, nor was the thickness of the mucosa taken into account because of its variability through ulceration and pseudopolyp formation.

Similar measurements were made of the various layers of the sigmoid colon in 20 additional specimens that had been resected for chronic ulcerative colitis but in which there were no strictures. Furthermore, 12 specimens of "normal" sigmoid colon that had been resected for carcinoma were measured. In none of these specimens was there complete obstruction, but the gut above the tumor was distended to a moderate degree in seven.

Results

The median measurements of the thickness of the various coats of the gut wall in each group are shown in Table 1. The most important differences were found in the thicknesses of the muscularis mucosae. Its median measurement in the 12 normal colons both above and below the carcinoma was 0.02 mm. In the 20 colons resected for ulcerative colitis without stricture, the median was 0.09 mm. In the 19 specimens of resected colon with ulcerative colitis and stricture the median figure for the zone not involved by stricture was 0.4 mm whereas that of the strictures was 0.8 mm. The ratio of the inner circular muscle of the muscularis propria to the muscularis mucosae was 75:1 in the normal and 1.8:1.0 in the stenotic zone of ulcerative colitis.

The measurements of the thickness of the contracted inner circular coat of the muscularis propria, on the other hand, were virtually the same in each group, and were similar to the measurements obtained in specimens of sigmoid colon in 10 normal subjects by Torsoli et al. Our range of 0.6 to 1.95 mm in the "normal" empty contracted sigmoid colon was slightly greater than theirs of 0.69 to 1.75 mm.

Measurements of the submucosal thicknesses in each group were also found to approximate closely.

TABLE 1. *Median Values and Ranges of Thickness of Inner Circular Muscle and Muscularis Mucosae.*

	No. of Specimens	Thickness of Inner Circular Muscle (Mm)		Thickness of Muscularis Mucosae (Mm)		Ratio of Inner Circular Muscle to Muscularis Mucosae
		Range	Median	Range	Median	
"Normal" colon*	12					
Above carcinoma		0.4-1.5	1	0.02-0.05	0.02	
Below carcinoma		0.6-1.95	1.5	0.02-0.05	0.02	75.0
Ulcerative colitis:						
Without strictures	20	0.4-2.1	1.4	0.02-0.43	0.09	15.5
With strictures	19					
Zone not involved by stricture		0.5-2.0	1.4	0.1 -1.6	0.4	3.5
Zone involved by stricture		0.7-2.7	1.5	0.5 -2.3	0.8	1.8

*Patients without ulcerative colitis.

Pathology of Stricture Formation

The muscularis mucosae, like the main muscle coat of the intestine, has two layers, an inner circular and an outer longitudinal, which in ordinary preparations of the normal colon are not readily delineated. The thickness of the muscularis mucosae seems to be

unaltered by contraction of the empty normal gut, merely being thrown into folds with the mucosa. In ulcerative colitis, however, the fibers of the muscularis mucosae are hypertrophied and seem to be in a state of contraction. Because of this, the inner circular layer of fibers can often be distinguished from the outer longitudinal. This contraction may be so forceful and sustained that the two layers become separated, thereby reducing the diameter of the lumen. This is most marked in the zones of stricture. Fibrosis is usually minimal and insufficient to account for the stricture.

Clinical Features

There were 13 females and six males. The age at the time the stricture was recognized varied from 15 to 60 years. The colitis involved the entire colon and rectum, with the exception of three cases in which only the left side of the colon was involved. The site of the stricture was either the sigmoid colon or the rectum (or both) in 14 cases, and in five the transverse colon. No strictures were found in the right side of the colon, in marked contrast to carcinomatous strictures in ulcerative colitis. The disease had been present for periods varying from five to 25 years—in the majority around 10 years. It was not possible to estimate the duration of the strictures. In all cases the disease had been continuous, without remission.

The following cases are described to illustrate the clinical problem.

Case 1. S.W., an 18-year-old nurse in whom the disease had begun insidiously at the age of 8, had become accustomed to a life of 4 to 8 loose stools per day. From time to time there had been an exacerbation, with urgent defecation, incontinence and almost continuous diarrhea day and night. Despite this she had grown normally and maintained a reasonable weight. Treatment had been spasmodic, and she had never been given salicylazosulfapyridine or corticosteroid therapy. There were no anal or systemic complications. For the past year she had had increasingly severe colicky pain in the middle and left side of the abdomen, and the stools remained loose.

When first seen in August, 1966, she weighed 57.2 kg (126 pounds) and looked relatively well. Apart from some mild left-sided abdominal tenderness, there was nothing abnormal on examination. Sigmoidoscopy revealed the classic appearance of mild ulcerative colitis. The rectum was of normal caliber up to the rectosigmoid junction, where it narrowed very appreciably to 1.5 cm. Barium-enema study disclosed a long stricture of the sigmoid colon. The entire colon was shortened, and the cecum contracted and practically continuous with the dilated terminal ileum. Colonic cytology did not reveal carcinoma cells.

On August 30, 1966, total colectomy and ileorectal anastomosis was carried out. The appearance of the resected specimen was that of chronic ulcerative colitis, with a stricture involving the full length of the sigmoid colon. Since operation the patient has been well. The rectum remains abnormal.

Case 2. V. H., a 41-year-old housewife, had a 10-year history of continuous ulcerative colitis involving only the left side of the colon and rectum. When she was first seen in July, 1967, her main complaints were episodes of severe colicky pain preceding defecation and the need to defecate several times daily. She looked well, and her weight was satisfactory. On examination feces could be palpated in the ascending colon. She had no local or systemic complications of ulcerative colitis. Sigmoidoscopy revealed active disease and a rectal stricture. Barium-enema examination demonstrated a marked rectosigmoid stricture with formed stools in the ascending and transverse colon. The rectum, descending colon and distal transverse colon were removed, and a mid-transverse colostomy was established. There was no evidence of carcinoma. The appearance of the bowel was that of chronic ulcerative colitis, with a stricture in the sigmoid colon. A year later she remained well, with no evidence of recurrence of colitis in the remaining colon.

Discussion

This study indicates that the essential factor in benign stricture formation in ulcerative colitis is the thickness of the muscularis mucosae. Stricture formation in ulcerative colitis appears to be a more intensive local manifestation of a process involving the whole colon.

Our findings contrast markedly with those of some authors, who emphasize fibrosis as the dominant feature in stricture formation in this disease. Warren and Sommers were the first to point out the importance of hypertrophy of the muscularis mucosae although they too emphasize fibrosis. Lennard-Jones et al. regard the striking shortening of the colon that is a feature of ulcerative colitis as being the result of a muscle abnormality and rarely of fibrosis.

Hypertrophy and thickening of the muscularis mucosae occurs in granulomatous colitis (Crohn's colitis) as well as in ulcerative colitis. In this disorder, however, the entire bowel wall is inflamed, and in areas of stricture, fibrosis extends through to the subserosa. Fibrosis of this extent never occurs in ulcerative colitis even when there is deep ulceration with destruction of the muscularis mucosae and submucosa. The strictures of ischemic origin are even more fibrotic, and the mucosa and submucosa become replaced by dense cicatrizing granulation tissue.

In this series strictures appeared only when the disease ran a continuous course without remissions and only after the disease had been present for five years. Edwards and Truelove also stress the continuous course, but one third of their cases were diagnosed within five years of the onset of the disease.

The site of stricture formation is mainly in the sigmoid colon and rectum, and in particular in the rectosigmoid and distal sigmoid areas. The reason for this is not clear. The lumen in the normal sigmoid colon has an appreciably smaller diameter and a higher motor activity than other areas of the colon. However, increased motor activity is related to muscular activity and does not seem to have relevance to the site of stricture formation in ulcerative colitis. Furthermore, in ulcerative colitis motor activity is markedly reduced.

The absence of benign strictures in the right half of the colon is clinically important, indicating that a stricture appearing in this region in a patient with ulcerative colitis is almost certain to be malignant in origin.

Our observation that stricture formation in ulcerative colitis is due mainly to the contraction and hypertrophy of the muscularis mucosae leads us to consider that such strictures are potentially reversible. It has been reported that radiologic strictures have disappeared when reviewed later.

The exact functions of the muscularis mucosae are still not understood, and further study should prove profitable. It is alleged that in the colon the dominant nervous influence is intrinsic sympathetic activity. What effect this has on the muscularis mucosae is unknown. A therapeutic agent capable of causing its relaxation might aid the reversibility of the pathologic changes that it undergoes and help distinguish benign from malignant strictures.

(The figures and references may be seen in the original article.)

THE USE OF HEPARIN*

Daniel Deykin, MD, New Eng J Med 280(17):937–938, April 24, 1969.

Although heparin is a widely accepted drug, there remains much confusion over its mode of action and over preferred routes of administration, dosage and regulation of therapy.

Heparin, a potent organic acid, is a mixture of sulfate-containing mucopolysaccharides, with molecular weights ranging between 8,000 and 15,000. Because of its strong electrostatic charge, heparin combines with a wide variety of proteins. Its anticoagulant action is a direct result of this property. It prevents the activation of clotting factor IX (PTC or Christmas factor), an essential component of the intrinsic coagulation mechanism. Furthermore, in conjunction with a plasma cofactor, it inhibits the action of thrombin. As a result, the thrombin-mediated conversion of fibrinogen to fibrin is retarded, and thrombin-induced aggregation of platelets may be prevented at high concentrations of heparin.

The exact routes of elimination of exogenously administered heparin are not precisely known. Approximately 20 percent can be recovered in the urine, and the remainder is apparently degraded in the liver by enzymes that presumably first remove the sulfate moiety and then degrade the saccharide. Heparin does not cross the placenta, and it does not appear in milk.

In addition to its anticoagulant action, heparin activates endogenous lipoprotein lipase. It also interferes with aldosterone elaboration. When given in doses of more than 10,000 units per day over a prolonged period (at least a year), heparin induces a severe metabolic bone disease characterized by os-

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teoporosis and collapse of vertebral bodies. Rarely, alopecia has been caused by heparin. More frequently, although the occurrence is still uncommon, transient thrombocytopenia may follow heparin administration.

There are significant differences between heparin and the coumarin congeners. Heparin is a direct anticoagulant. Its action is a reflection of its chemical properties, and it is immediately effective in vitro as well as in vivo. The coumarins act only in the liver, where they retard the synthesis of certain procoagulants; therefore, the action of these drugs is manifest only in vivo and after an appreciable delay. The coumarin agents do traverse the placenta and are found in the milk.

The rationale for the use of heparin derives exclusively from its ability to block the coagulation mechanism. A current formulation of the pathogenesis of thrombosis recognizes three distinct types of thrombosis. The arterial or white thrombus arises primarily through the interaction of circulating blood platelets with an injured or abnormal vessel wall. The coagulation sequence itself has only a limited role in the genesis of this platelet-mediated thrombus. In contrast, the red thrombus that forms in columns of static blood arises through activation of the blood-clotting mechanism, and platelet reactions are of secondary importance. Similarly, activation of the clotting sequence is directly involved in the pathogenesis of disseminated intravascular coagulation, or consumption coagulopathy. These observations suggest that anticoagulant therapy in general, and heparin specifically, will be effective in preventing stasis thrombi and disseminated intravascular coagulation, but will have limited benefit in preventing the platelet nidus that triggers arterial thrombosis. Accordingly, the most uniformly accepted indications for heparin therapy are deep thrombophlebitis, acute pulmonary embolism and disseminated intravascular coagulation. Heparin also is frequently used in the management of acute arterial occlusion of the extremities to prevent distal propagation of stasis thrombus before operation. Finally, heparin is added to blood during extracorporeal circulation to prevent clotting as the blood flows over foreign surfaces.

Heparin therapy is prophylactic. There is no convincing evidence that heparin has any effect on preformed thrombi except to prevent distal propagation and to limit thrombin-mediated platelet accretion on the surface of the thrombus. Accordingly, the resolution of vascular occlusion that often occurs during heparin therapy reflects primarily endogenous mechanisms—thrombolysis, thrombus fragmentation, organization and recanalization and development of collaterals—rather than direct action of heparin. The demonstrated efficacy of heparin in pulmonary embolic disease is due to prevention of further thrombus formation, while lysis of the embolus and organization of the seeding thrombus are in process.

It has been postulated, on the basis of experimentally induced pulmonary emboli in animals, that heparin may have an additional action in the treatment of pulmonary embolism. At high concentrations it blocks thrombin-mediated release of pharmacologically active amines from platelets. It may thereby diminish the bronchospasm and dyspnea that accompany pulmonary embolism.

Heparin must be administered parenterally. Since they are frequently associated with considerable bleeding into the injection site, intramuscular injections should be avoided. Heparin is usually given subcutaneously or by intermittent intravenous infusion. The advantages of the intravenous route are the immediate onset of action, the ease of controlling the dose and the rapidity of dissipation of the anticoagulant effect after the drug is discontinued. Furthermore, some investigators believe that the dramatic interference with the coagulation mechanism that immediately follows the intravenous injection may be the major therapeutic action of heparin. The advantages of subcutaneous injections are the relative ease of administration and the avoidance of indwelling catheters and needles. However, the absorption of the drug is variable, and neutralization of a subcutaneously administered dose is clearly more difficult than neutralization of one given intravenously.

There is no uniformity of opinion concerning the optimal dose of heparin, the frequency of administration or the method of regulating the dose. In one widely adopted schedule heparin is administered intravenously at four-hour intervals in amounts (approximately 5,000 units) sufficient to maintain the whole-blood clotting time at twice the control values, three and a half to four hours after each injection. Others suggest that during the initial 48 hours of therapy, larger doses (10,000 to 15,000 units) be given intravenously at four-hour intervals without regard to whole-blood clotting times, and that the dose subsequently be reduced to lower levels (usually 5,000 units). Still others advocate a standard dose between 5,000 and 10,000 units intravenously every four hours, with no attempt to monitor the whole-blood clotting time. There is equal variation in schedules for subcutaneous administra-

tion, but many clinics have adopted a regimen of 15,000 to 20,000 units every 12 hours.

Although several experimental models have been offered to substantiate one or another regimen, enthusiasm for a given scheme is largely anecdotal, for no clinical data available establish the superiority of any schedule or even the route of administration. It is clear, however, that the response to a given dose of heparin may be quite variable. Early in the course of florid thrombophlebitis or massive pulmonary embolism, a large dose may have little effect on the clotting time, but as the acute process subsides, sensitivity to heparin is restored. Therefore, a standard dose may be insufficient early in the course of therapy and excessive later. The major objections to adjusting the dose of heparin in response to frequent determinations of the whole-blood clotting time are the imprecision of the assay as it is usually performed and the excessive time required to perform the test. Several clinics have reported that the activated partial thromboplastin time, a relatively simple and rapid test, can be successfully employed to monitor heparin therapy. If the assay is more widely adopted, more uniform dose schedules may yet emerge.

More frequent bleeding occurs during heparin therapy than with the oral agents, for heparin is a more potent anticoagulant. Although there is no direct correlation between the dose of heparin and the rate of occurrence of bleeding, more frequent bleeding follows sustained intravenous administration of high doses every four hours than lower dose schedules. The specific antidote to heparin is protamine, a highly basic protein, which forms an irreversible complex with heparin. A useful schedule for neutralizing an intravenous dose of heparin is to administer 1 mg of protamine for every 100 units of heparin given at the last injection. Unfortunately, a gross excess of protamine may prolong clotting. Therefore, the neutralization of a subcutaneous injection of heparin must be carried out with smaller doses of protamine given at intervals.

In many cases bleeding during heparin therapy heralds a previously unsuspected lesion, but too frequently bleeding reflects errors in management. A deep intramuscular injection of some other medication may cause profound bleeding in a heparinized patient, even if the injection occurred several days before the initiation of heparin therapy. Furthermore, agents that interfere with platelet aggregation reactions, which are the primary hemostatic defense in heparinized patients, may induce bleeding. Acetylsalicylic acid is such an agent; it should be scrupulously avoided in patients receiving heparin.

Heparin is a potent drug. Its toxic effect, bleeding, is a direct extension of its therapeutic action. Given judiciously, it remains the most effective antithrombotic agent available.

PRINCIPLES OF TREATMENT AND SPECIAL PROBLEMS IN VASCULAR TRAUMA

Max R. Gaspar, MD FACS, Richard L. Treiman, MD FACS†, J. Howard Payne, MD FACS*, Philip D. Rothschild, MD†, and Donald J. Gaspard, MD‡, Surg Clin N Amer 48(6):1355–1364, December 1968.*

Surgeons who treat trauma face an increasing incidence of vascular injury and are obligated to apply the knowledge which has been gained in recent years. Optimal treatment not only saves lives but also saves limbs and in addition preserves their function. The need for vascular repair was evident in World War I. Still in World War II only 81 of 2,471 patients with arterial injuries had suture repair of the injury, and in only three of these patients was an end-to-end anastomosis accomplished. At that time the techniques of vascular surgery were not developed sufficiently to be useful. In the Korean and Vietnam conflicts, vascular repair has been used with increasing frequency. Civilian experience has paralleled military experience.

At the Los Angeles County—University of Southern California Medical Center during the past 20 years, over 400 acute vascular injuries have been treated. This experience has been under constant review and has resulted in the development of important principles which have led to gradual improve-

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ment in the management of these injuries. These principles are (1) prompt recognition, (2) prompt arteriography, (3) rejection of the diagnosis of arterial spasm, (4) prompt precise repair, (5) proximal and distal thrombectomy, (6) intraoperative arteriography, and (7) prompt reoperation if arterial insufficiency recurs after repair.

Prompt Recognition

The recognition of major vascular trauma in a wound from which blood is spurting is simple, but recognition of major vascular trauma in the chest, abdomen, and in the extremity which is not bleeding visibly, and particularly when there has been blunt trauma, is not always easy. Any patient who is in shock or who has a penetrating injury or severe blunt trauma, and especially a fracture, must be considered to have a vascular injury until proven otherwise. Diagnosis usually may be made by physical examination. In the extremities a check must be made for the five P's—pain, pallor, paresthesia, paralysis, and pulselessness. Any or all of these may be absent or misleading even though a major vascular injury is present. When a bullet or stab wound does not appear to be in the vicinity of a major vessel, it is easy to overlook the possibility of major vascular damage. A bullet need pass only within a few centimeters of a vessel in order to contuse it and cause thrombosis. Pulses may be present under these circumstances, particularly on the initial examination, only to be absent at a subsequent examination. In one of our studies ten patients presented at the hospital with a palpable peripheral pulse despite the presence of proximal arterial injury. These were side-wall lacerations of the vessel which allowed distal circulation. Prompt recognition demands a high index of suspicion when the patient is first seen and throughout his course.

A hematoma in the vicinity of a wound must be observed closely, for it usually denotes a lateral injury to a vessel. Steady expansion of a hematoma demands exploration even though the patient may not be in shock and a distal pulse may be present. Even though large, a hematoma seldom produces sufficient compression of an artery to cause loss of a distal pulse unless the patient's pulse pressure is low as a result of hemorrhage or for other reasons. A pulsating hematoma denotes a false aneurysm and must be operated upon. In most cases, over or near the false aneurysm there is a systolic bruit which occasionally is carried into the diastolic phase.

Secondary hemorrhage from a wound is an ominous sign. It has been called the "red signal" presaging a final massive hemorrhage. In World War II in the China-Burma-India Theater, Freeman noted final massive hemorrhage usually to occur between the tenth and sixteenth days, but as early as two days and as late as three months after injury. In all instances there had been massive hemorrhage or recurrent bleeding in the forward area and the patients were admitted with severe anemia.

In trauma to the chest one must be aware of the possibility of injury to the aorta and its major branches, the innominate, carotid, and subclavian arteries. Shock is not always present. In one of our patients who had sustained blunt trauma to the chest there was complete transection of the intima of the aorta with only a small posterior disruption of the media and adventitia. The patient was not hypotensive but the diagnosis was suspected because of a moderately widened mediastinum noted on x-ray of the chest. As in most instances of aortic injury accompanying blunt chest trauma, the disruption occurred just distal to the left subclavian artery. There was no alteration in the pulses of the upper extremity but a systolic bruit developed over the anterior and posterior chest walls.

Blunt trauma to the abdomen may cause particularly difficult diagnostic problems. The principal findings are shock and hemoperitoneum. The latter may be confirmed by peritoneal tap. Retroperitoneal hemorrhage is particularly difficult to diagnose and requires frequent careful examinations of the patient, repeated hemoglobin or hematocrit determinations, abdominal roentgenograms to determine psoas shadows, and intravenous pyelography.

Prompt Arteriography

Arteriography is unnecessary and undesirable in the massively bleeding open wound. However, it is of great value when the signs of arterial injury are indefinite, particularly in blunt trauma. This is especially true with fractures.

Arteriograms of the aorta and upper extremity vessels are obtained by catheterization of the femoral artery according to the Seldinger technique. Lower extremity arteriograms are performed by direct needle puncture into the femoral artery. In cases of fracture it is often expedient to do them on the operating table preliminary to operation.

By following the principle of prompt arteriography we have disclosed instances of arterial trauma which were only slightly suspected. Our experience is not in agreement with that of Lumpkin and his associates and Patman and his associates who noted

that extravasation of the contrast material does not occur in all cases of loss of integrity of the artery.

Rejection of the Diagnosis of Arterial Spasm

It cannot be denied that arteries may go into spasm because of their intrinsic musculature, but this is a dangerous diagnosis to make when there is trauma. Spasm is seen most frequently in the postoperative period after major vessels have been clamped for a prolonged period of time and also when vessels are stretched in their longitudinal axis. Spasm is seldom seen in a traumatized artery, not even in the vicinity of the application of an occluding clamp. In trauma there may be unrecognized factors which produce spasm of blood vessels but, if so, they seem to affect small vessels such as those of the skin, but not the larger arteries.

It is convenient but hazardous for the surgeon who does not feel pulses in an extremity distal to an injury or who notes pallor and coolness to conclude that these findings are due to arterial spasm. This is particularly true in cases of blunt trauma or when a bullet wound or stab wound is not adjacent to a major vessel. Spasm also may be diagnosed with an arteriogram, particularly in cases of blunt trauma, but in our experience there has been contusion with thrombosis or intimal disruption in all such cases rather than spasm.

For these reasons we reject the diagnosis of arterial spasm in trauma and insist that all patients in whom such a diagnosis is considered possible must be operated upon. A questionable pulse must be considered an absent pulse.

Prompt Precise Repair

Once the diagnosis of vascular trauma is made, there should be no delay in getting the patient to the operating room. When there is massive hemorrhage, the need for prompt repair is obvious. When hemorrhage is not a compelling factor, delay will result in ischemia of the part distal to the injury.

Proximal and distal control is the prime fundamental of vascular repair. Often this means an incision placed in uninjured areas proximal or distal to the site of injury rather than exploration through the wound. A retroperitoneal approach to the external iliac artery when there is an injury in the common femoral artery is an example of such an approach.

After bleeding is controlled by proximal and distal occlusion of the vessel, the vessel is elevated from its bed in the region of the injury and cleaned of surrounding tissue. We do not advocate stripping the adventitia from an artery, as it is possible to remove too much adventitia and leave a vessel without structural integrity. Contused ends of severed arteries should be carefully trimmed unless the injury has been a severance or laceration with a sharp object such as a knife or glass. In high-velocity gunshot wounds it is particularly important to excise approximately 1 cm of artery on each side of the wound because necrosis from the blast effect is always greater than is grossly visible.

When the ends of an artery can be brought together without tension an end-to-end anastomosis is preferable. This is constructed with precise over-and-over arterial sutures of fine silk taken close to the cut edge and at approximately 1 to 2 mm intervals. Triangulation according to the technique of Carrel is useful in the smaller arteries. Interrupted sutures are seldom necessary in vessels larger than 3 mm in diameter. If it appears that there may be some narrowing at the site of the anastomosis, this may be overcome by one of five methods: (1) The ends of the artery are quite easily dilated by spreading them gently with a blunt-nosed hemostat. This is highly recommended in all but the largest vessels. (2) The cut ends may be cut on the bias but, in making such an angled anastomosis, one must be sure that the artery is in accurate axial rotation. Otherwise there may be as much as a 360-degree twist which would produce obstruction. (3) An alternate method is to spatulate the two ends by cutting longitudinally each vessel at opposite poles and trimming the edges. Here, too, one must be careful not to rotate the vessel. (4) The fourth method is to perform the anastomosis over a catheter. This is particularly useful in the internal carotid artery where the catheter is used as an internal shunt to maintain flow to the brain. The catheter may be introduced through the cut ends and brought out prior to the last few closing sutures of the anastomosis or it may be introduced through a proximal or distal arteriotomy from which it is removed after the anastomosis is completed. (5) A vein patch may also be used in conjunction with end-to-end anastomosis. Half of the anastomosis is completed and the patch is then used to close the remaining defect.

Lateral suture repair is feasible only in the occasional case in which there has been a clean stab wound. In gunshot wounds in which there is loss of substance of one side of an artery, it is best to resect the injured area and to perform an end-to-end anastomosis. Occasionally in small lateral gunshot wounds, it is possible to debride the edges of the

wound and apply a patch graft. Vein patch grafts are preferred to prosthetic grafts because of the danger of infection in traumatic wounds. Under these circumstances foreign bodies of a cloth nature are apt to produce prolonged drainage and secondary hemorrhage and thrombosis.

When a segment of artery has been destroyed, attempts should be made to replace it with another conduit. If the saphenous vein is available from the injured extremity or the opposite lower extremity, it should be reversed and used as a graft using an end-to-end anastomosis proximally and distally. Occasionally it has been possible to remove the hypogastric artery to bridge a short segment of lost artery. Also it has been possible to excise the external iliac artery and to replace it with a Dacron graft and use the excised external iliac artery as a graft to replace the segment of artery lost by trauma. Prosthetic grafts are avoided in all but desperate situations because of the danger of infection.

Proximal and Distal Thrombectomy

Antegrade bleeding and retrograde bleeding have long been considered adequate evidence of patency of the proximal and distal arterial tree prior to final closure of an arterial repair. However, such bleeding can occur in the presence of nonoccluding clots either proximally or distally but especially in the distal arterial segment. We have therefore adopted the practice of passing a Fogarty catheter proximally and distally prior to placing the few final sutures. It has been surprising how much clot has been removed on occasion. Such clot undoubtedly would contribute to postoperative thrombosis and ischemia.

The balloon catheter must be used gently because it can penetrate the normal arterial wall, producing hemorrhage and arteriovenous fistula, and it can remove circumferential segments of intima from an artery. The balloon of a Fogarty catheter must be distended only sufficiently to contact the arterial walls but not enough to distend the artery. This fine degree of touch can be appreciated by the operator who simultaneously controls the withdrawal of the catheter and the diameter of the balloon by variation of finger pressure on the syringe connected to the balloon. If the Fogarty catheter distends the vessel wall, the artery is stretched as the catheter is withdrawn and this is one of the few mechanisms which may produce intense arterial spasm.

Intraoperative Arteriography

We insist that arteriography be performed in all cases after completion of the repair. On several occasions we have demonstrated small clots within the arterial lumen or actual complete occlusion. Even more importantly, we have demonstrated additional unrecognized injuries to the artery.

Prompt Reoperation When Necessary

The patient must be observed closely at frequent intervals in the postoperative period. The fingers and toes should be exposed if possible so that their color and temperature and their capillary filling can be assessed. Capillary filling generally is a poor sign of arterial patency, being valid only when it is unequivocally prompt and full. Ideally bandages and casts should be placed in such a way that peripheral pulses can be palpated. Circular bandages should not be applied to extremities because they almost inevitably result in constriction. It is well to mark with a pen the site of peripheral pulses so that nurses, interns, and residents unacquainted with the patient can readily feel in the correct place for pulses. If pulses disappear at any time in the postoperative period, an arteriogram should be performed only if there is doubt that the artery is occluded and serious consideration should be given to returning the patient to the operating room for re-exploration of the arterial wound. This is often a difficult decision when the patient is extremely ill and in the presence of infection, but loss of arterial continuity is tantamount to amputation. The wound should be re-explored unless the operation would jeopardize the patient's life.

Special Problems

Vascular Injuries of the Upper Extremity and Neck

Exposure of the innominate, carotid, subclavian, and axillary vessels presents the greatest difficulty. For exposure of the great vessels arising from the arch of the aorta, we prefer a sternum-splitting incision including resection of the proximal portion of the clavicle subperiosteally if necessary. In the case of the axillary artery an S-shaped incision is made, centered over the axilla. The tendon of the pectoralis major muscle may be transected for exposure of the proximal portion of the vessel. Exposure of the arteries in the neck, arm, and forearm pose no major problem.

In World War II when acute arterial injuries were managed by ligation, the incidence of gangrene of the lower extremity was twice that of the upper extremity. Ziperman, in reporting Korean War experience, noted gangrene of the arm requiring amputation in less than 10 percent of patients with brachial

artery ligation, whereas 50 percent of patients with ligation of the femoral artery developed gangrene requiring amputation of the leg. We have been surprised in reviewing upper extremity arterial injuries to learn how well patients have fared following ligation. No patient has complained of postoperative claudication preventing performance of his usual work. Nevertheless, we strongly advise arterial repair of major arteries. The radial and ulnar arteries need not be repaired except in those instances of complete or nearly complete severance of the forearm or wrist.

Common carotid and internal carotid arteries should be repaired if possible because of the possibility of arteriosclerotic carotid artery stenosis on the opposite side in later life. We have repaired a completely transected internal carotid artery four hours after injury. The external carotid and vertebral arteries need not be repaired but merely ligated.

Injuries of the Thoracic Aorta

Gunshot wounds and stab wounds of the thoracic aorta are apt to produce massive hemorrhage and rapid death, but we have seen a stab wound of the thoracic aorta which sealed spontaneously. Blunt chest trauma characteristically produces an injury immediately distal to the subclavian artery at the location of the ligamentum arteriosum. Hemothorax and a widened mediastinal shadow on x-ray are the principal signs of thoracic aortic trauma.

There should be no delay in operating upon patients with suspected injury to the thoracic aorta. If the vital signs are stable and there is doubt about the diagnosis, retrograde aortography should be performed early. We have seen a young man who did not have an aortogram until the ninth day after injury at which time a complete intimal transection was demonstrated. The operation was difficult because of the extensive tissue reaction which would not have been present in the first few hours after injury.

If at all possible, the patient should be transferred to a center where bypass procedures can be performed. In the case of descending thoracic aorta lesions, only left heart bypass is needed. Under desperate circumstances it is permissible to explore the chest in an effort to stop hemorrhage even if ideal conditions are not present, but this should be done only if the diagnosis is certain or strongly suspected.

Injuries to Major Veins and Arteriovenous Fistules

In a previous report we noted five of 52 patients with major venous injury who died as a direct result of their venous injury. In addition eight patients developed arteriovenous fistulas because surgical exploration was not attempted. Major venous injuries therefore cannot be dismissed lightly. When there is profound shock, evidence of massive bleeding in a wound, an expanding hematoma, or a to-and-fro bruit, exploration should be done without delay. Ligation is permissible in most circumstances, but lateral suture repair should be done in larger veins, including the popliteal vein, if feasible. Grafts to bridge venous defects are not advocated. Postoperative anticoagulants are of little value and postoperative thrombophlebitis has not been a major problem after repair or ligation.

Injuries of the vena cava are of special interest. If the patient survives long enough to be operated upon, the vena cava should be repaired by lateral sutures rather than by ligation, and aorta-vena caval fistulas should be repaired. Five of eight patients in our series survived.

Intra-abdominal Vascular Trauma

Arterial and venous injuries within the abdomen are seldom isolated injuries. Only the major vessels such as the aorta, vena cava, iliac arteries and veins, celiac axis, and hepatic and renal arteries need be repaired. The splenic, inferior mesenteric, and hypogastric vessels may be ligated.

The renal arteries are of special importance because hypertension may result from injury. We have seen hypertension develop within ten days of blunt trauma to a renal artery. In cases of massive retroperitoneal hemorrhage the aorta, vena cava, and renal arteries should be exposed to be sure they are not injured. If they are intact a prolonged search for another bleeding source is not necessary unless bleeding is profuse. Early repair of a renal artery is more apt to be successful than late repair, at which time the kidney may have to be sacrificed.

The iliac veins may bleed profusely and cause exsanguination due to a fracture of the pelvis. The surgeon should not hesitate to operate when the patient with a fracture of the pelvis is in shock and has signs of a retroperitoneal mass.

Arterial Trauma Due to Fractures and Dislocations

The reason for generally poor results in fracture-associated arterial injuries is procrastination and a failure to appreciate the signs and symptoms of arterial insufficiency. In a group of 23 patients with fracture-associated arterial injuries, major amputation was necessary in 18 patients. A high index of suspicion, an aggressive approach, and application of the

principles advocated in this report could have prevented the loss of these limbs.

Dislocations of the knee nearly always result in injury to the popliteal artery and subsequent amputation unless this artery is exposed and repaired. In this injury in particular, there is apt to be contusion of the artery without transection but with intimal disruption and consequent occlusion and thrombosis. We have seen this same lesion in the common iliac artery in severe blunt trauma. We have improved our own results only by insisting that the popliteal artery be explored in all dislocations of the knee.

When internal fixation of the bone or joint is necessary, it is important to expedite this portion of the operation to avoid prolonged ischemia. We have seen one instance in which a meticulous bone repair lasting several hours jeopardized the viability of the extremity. However, it is best to stabilize the bone before repairing the artery so as not to disrupt the arterial repair during bone manipulation.

Ancillary Measures

Blood and its substitutes must be used as necessary to combat shock, but should not be given continuously when it is more important to stop the source of hemorrhage. Occasionally it is better to operate upon the patient in shock in order to secure the bleeding vessel. In the immediate postoperative period the blood volume must be maintained in order to help prevent stasis in the arteries distal to the repair.

Sympathetic block, sympathectomy, and vasodilating drugs have had little place in our practice. We have not been able to demonstrate any benefit from using them. Local and systemic papaverine has theoretical appeal, but there is seldom spasm in arterial trauma. When vessels are small for anastomosis they should be dilated with a blunt-nosed hemostat.

Heparin is of value during the time that an artery is clamped, but the danger of bleeding into the wound precludes its use in the postoperative period. Heparin will not salvage a poorly performed repair nor the vessel filled with clot which has not been removed by proper use of the Fogarty catheter.

Fasciotomy in the upper and lower extremities often saves an extremity. It should be done in cases in which there has been prolonged ischemia or in which major veins were ligated or in which swelling of the extremity is already present. It may be performed through a very small skin incision, although the fascial incision can be several inches in length.

When distal pulses have been restored in an extremity, that extremity should be elevated in an effort to prevent swelling. Elastic wrappings are also permissible if they are not constrictive and do not obscure the palpable pulses.

Summary

The techniques of vascular repair have been developed to the point where the surgeon dealing with trauma should apply certain principles which will result in improved results. These are (1) prompt recognition, (2) prompt arteriography, (3) rejection of the diagnosis of arterial spasm, (4) prompt precise repair, (5) proximal and distal thrombectomy, (6) intraoperative arteriography, and (7) prompt reoperation when necessary.

A number of problems relative to vascular trauma require special knowledge and technique. Several ancillary measures are of aid in improving results.

(The references may be seen in the original article.)

SINGLE AND MULTIPLE ATTACKS OF TRANSMURAL MYOCARDIAL INFARCTION

RELATIONSHIP TO AGE, MORTALITY, AND SURVIVAL

Arthur M. Master, MD, and Richard P. Lasser, MD,
JAMA 209(5):672–675, August 4, 1969.

We dealt with the age and prognosis of 484 persons experiencing a first attack of true transmural myocardial infarction who survived long enough to be hospitalized and have an electrocardiogram. One hundred and one patients experienced two attacks, and 15 patients had three attacks. The third attack proved fatal in every instance and death was fairly sudden. The attrition rate for the initial attack was 21.3% for the

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first three months, 6.6% for the next nine, 5% for the second year, but only 2% per year between the second and 12th year. For the 101 patients who had a second attack, 39% died within three months, 47.7% after one year, and 73.5% within five years. There is not, and cannot be, an accurate death rate for first attacks in which the patient is hospitalized, since it does not include the more than 40% dead before any possible medical treatment. However, the prognosis after recovery from the first attack was accurate since all patients were observed.

Since coronary heart disease is the leading cause of mortality and morbidity in this country, it is not surprising that there have been many reports dealing with mortality and survivorship after myocardial infarction. Considerable variation in prognosis can be noted among these various reports. With increasing knowledge of the natural history of coronary disease it has become apparent that variation in death rates results both from the type of patient included in the study and even more significantly, from the length of time that patients are seen after the infarct. With regard to the type of case included, studies have included all kinds of "coronary" attacks ranging from the most mild, to patchy or diffuse subendocardial infarction, to the complete transmural infarct.

The current study will be concerned only with complete transmural infarction which in general is far more serious than the more mild attacks of diffuse necrosis or subendocardial infarction. Unfortunately, the vast majority of reports have lumped transmural and subendocardial infarcts together. As far as length of time elapsed after attack is concerned, our study will encompass patients as they are seen by the practicing physician or cardiac consultant, ie, at somewhat random times after infarction. Thus, the prognosis of the first attack of transmural infarction as reported in this paper would not be comparable to complete data gathered in epidemiological investigations. In this communication we will have missed those patients experiencing sudden death without a preceding symptomatic interval, those dying with symptoms but prior to arrival of medical attention, and those dying during transport to the hospital. This group of sudden and early deaths after coronary thrombosis constitutes more than 40% of the total number observed during the acute attack. Although all patients in this current report had been hospitalized and had had electrocardiographic evidence of transmural infarction, the values for immediate death rates must be regarded in the light we have just described. The long-range prognosis after recovery from the first acute transmural infarction, however, is not beset with the same uncertainties as the estimation of the mortality in the first attack since all patients in the current study were observed for the duration of the research. Hence, this investigation will present data on survival after the first attack of acute transmural infarction and also observations on all the second and third episodes in those who survived the initial attacks. The extremely high mortality, probably 100%, in the third episode will be stressed. We will describe many more variables than those encountered in the endeavor to obtain accurate figures in regard to mortality and survivorship. This is necessary to obtain a perspective of the field. Finally, we will present thoughts on possible means of prevention of death after the first attack of transmural infarction.

The data to be reported in this study were gathered from a consecutive series of 484 private patients who experienced a first transmural myocardial infarction. The patients were those of one of us (A.M.M.) and they were examined at least every six months after the initial attack. Others who were seen personally once or only a few times were nevertheless observed continuously by the family physician. A full-time research worker was employed to gather and classify the necessary data. The patients all showed the ECG pattern of acute transmural myocardial infarction with significant Q waves, RS-T segment elevations, and T-wave changes. The clinical pattern of transmural infarction was present in part or in toto, namely, severe chest pain, diaphoresis, fall in blood pressure, etc. In those who survived for several days or longer, the customary increase in temperature, white blood cell count, and serum enzyme levels were also present.

Type of Subject

There were 434 men and only 50 female patients, a ratio of 8.7 men to 1 woman. Manual workers and laborers were 15% of the total group; store workers, office workers, and businessmen (including executives) were 58.5% of the total. Professional persons, including physicians, were 14.6% (physicians, 6.9%) of the total, and housewives were 8.7% of the total. Those patients who were retired formed 3.2% of the entire group. These patients were largely middle-class or well-to-do people. The patients were white, and a majority was Jewish. Practically all were residents of the immediate metropolitan area of New York.

Of the total group of 484 patients who had suffered a myocardial infarction, 383 (79.1%) exper-

ienced only a single attack and 101 patients (20.9%) suffered two attacks of myocardial infarction. Fifteen patients were involved in three attacks.

The age of the male patients having the first attack of myocardial infarction ranged from 30 to 89 years. The peak age of attack in men was the 50- to 54-year-old age group (21.0%), the next highest incidence was the 45- to 49-year-old age group (18.7%). Thirty-eight percent of the men were less than 50 years of age. Only 12.7% were 65 years of age or more. The average age was 53.0 years.

In the women, the peak age of attack was between 55 and 59 years, and the second highest age grouping was the 50- to 54-year-old age group. Only 20.0% of the group fell under the age of 50, in contrast to 38.0% in men. The average age was 55.9 years.

The age distribution of men having a second myocardial infarction as compared with the first attack of these patients is shown. The peak attack age for second infarction was the 55- to 59-year-old age group and the average age was about five years more than in the first attack. Many second attacks had already occurred in the 35- to 39-year-old age group. We repeat, that in general, reliability of the ages in the first attack is not as good as that in the second. The latter is dependable since every patient who survived the first attack was observed. The average age of women suffering a second infarction was 61.0 years.

The mean age of those unfortunate enough to experience a third attack was only 54.7 years. All of the 15 third attacks proved fatal and death was often fairly sudden.

Immediate and Long-Term Survival

The survival rates were calculated by the life table method of Merrell and Shulman. This type of statistical analysis is particularly suited for evaluation of survival in long-term chronic diseases such as coronary artery disease, since it takes cognizance of the fact that patients enter the study at different times, are observed for varying periods, and a substantial number are still alive at the conclusion of the study. The use of a simple, direct calculation of the proportion of those living and the proportion of those dead after a given period would be misleading.

The survival curve for the 484 men and women in this series who experienced their first attack of transmural infarction is seen. During the first week after infarction, 63 patients died (13%), the majority of deaths occurring within the first two days. An additional 8.3% of these patients died during the three-month period after infarction. Thus, our total mortality in the first three months was 21.3% (78.7% surviving). At 1 year, 72.1% of the original group was alive; at 5 years, 57.6%; at 10 years, 48.3%; and at 15 years, 31.2%. The survival curve thus drops precipitously during the first three months and then much less so during the remainder of the first year and during the second year. After the second year, the curve becomes virtually a straight line and with but a slow descent, dropping the rate of death to about 2% per year to the 12th year. Then the rate falls more rapidly again, about 4% per year to the 15th year.

The comparison of men less than 50 years of age at the time of first attack in this series with those who were more than 50 years of age when the attack occurred is graphically shown. The mortality during the first three months was strikingly different with 91.5% of the men less than 50 years old surviving at three months compared with only 71.5% of the men more than 50 years of age. However, thereafter the rate of loss per year was about the same in both age groups and the curves therefore run generally parallel.

The survivorship of women proved to be somewhat less satisfactory than men. The small number of patients, however, makes the data less definite. A trend is indicated. The survival rate three months after the first attack was 71.5%, at one year, 68.8%, and at three years, 49.5%. These values were similar to those for men more than 50 years of age.

The survival rates for patients experiencing a second myocardial infarction (101 patients) is shown (Fig 4). Thirty-nine percent of these 101 patients died during the acute phase and in the first three months. Death was usually sudden. At one year after the attack, 52.3% were alive, at five years, 26.2%, and at eight years, 20%.

Comment

In this study we have attempted to describe the death rates observed in the private practice of cardiology in patients with true transmural infarction. By the very nature of patient selection, namely, the requirement that each patient manifest characteristic changes in the electrocardiogram, the subjects who do appear have all survived long enough to have an electrocardiogram made either at home or, in the vast majority of cases, upon arrival in the hospital. From epidemiological studies, it is known that about 40% of deaths from acute coronary thrombosis

occur prior to hospitalization (or the patients are dead on arrival), and thus, these patients do not appear in the current series, or in any series dealing with hospitalized patients. However, as the object of this study is to report on the prognosis of true transmural infarction, this type of patient selection is unavoidable. The reader should therefore clearly understand that the mortality during the acute period of the first episode, which was found by us to be 21%, may be far from the true figure. Arriving at the complete death rate in the first attack of coronary thrombosis is further complicated by the fact that patients who have "silent" attacks may be missed entirely and the diagnosis may be made only by chance at a later date when an electrocardiogram is perhaps made during a routine examination. Again, the diagnosis may be missed because diagnostic skill varies among physicians. Death rates in early reports, decades ago, cannot be strictly compared with current studies because of the great improvement in the diagnosis of coronary thrombosis. Furthermore, there is now more success in therapy, eg, in the treatment of arrhythmias. In 1920 and 1930 the diagnosis was uncertain and the disease was considered unusual. When the change was made from the three-lead and four-lead ECG to the current 12-lead ECG, recognition of transmural infarction improved considerably. Enzyme investigations have also aided in the diagnosis of the through and through infarct. This change occurred in the late 1940's.

The difficulty in nomenclature has already been stressed. One of us (A.M.M.) and his colleagues did make the distinction between transmural and subendocardial infarction, but in general, years ago, any change in the electrocardiogram was labeled coronary thrombosis or occlusion. Few, if any, distinguished between subendocardial and transmural infarction. Because of these problems in calculating the survival rate of myocardial infarction, the mortality during the first month after infarction has been variously reported as from 20% to 60%. Of the patients who survive the acute phase, prior studies indicate that from 55% to 80% can be expected to be alive 5 years later, 30% at 10 years, and 10% at 15 years.

These problems besetting the determination of death rate during the first attack do not apply to those who have survived this episode. Hence the mortality of those suffering second and third attacks are reliable since all were then observed completely with periodic examinations and electrocardiograms.

The more favorable prognosis in this series of subjects less than 50 years of age at the time of first infarction compared with older groups has been observed in other studies. However, this observation applies only to hospitalized patients since there is some evidence that sudden death due to coronary thrombosis occurs more frequently at the younger ages.

Women suffered myocardial infarction at a somewhat later age than men but in our series the difference was not great.

Third attacks all proved to be fatal. Death was nearly always sudden or in a very short time.

Death in acute myocardial infarction may be due to sudden ventricular tachycardia and fibrillation. So in practice we have made it a point that after the first transmural infarction, if there are more than rare ventricular premature beats, the patient is continuously treated with quinidine sulfate or procainamide hydrochloride. Possible causes of the arrhythmias are removed, eg, tobacco, liquor, coffee, tea, and other caffeine drinks. We also introduce preventive measures as gleaned from coronary profile studies. Definite hypertension is treated by appropriate therapy, hypercholesterolemia by diet and with new cholesterol-lowering agents. Weight is reduced in the obese. A serious attempt is made to engender a philosophic attitude in the tense. Cigarette smoking is interdicted. Work and physical activity in moderation are encouraged. For appropriate patients, graded exercise may be indicated. However, undue mental, emotional, and physical strain is to be avoided in those who are sick.

(The tables, figures and references may be seen in the original article.)

RESEARCH SECTION

SOME NAVY MEDICAL RESEARCH ACCOMPLISHMENTS

How do Navy men respond to stressful situations such as living in the isolated Antarctic or undergoing underwater demolition team operations or training? Under the aegis of Human Effectiveness Research at the Naval Medical Neuropsychiatric Research Unit (NMNPRU), San Diego, California, research is being conducted to better predict effectiveness under just such trying conditions. Data collected and analyzed include personal history information, both biographical and medical in nature; when these factors are combined with knowledge of the occupation and psychophysiological setting of each man, a fairly accurate prediction of his short-term response to stress can be made. The results of this research are thus aiding in the selection of personnel for stressful duty. Also being explored at the NMNPRU are the problems of general health change in different shipboard environments.

Extending their inquiry into the likelihood of effectiveness of a sailor's first enlistment, NMNPRU has designed and tested a prediction system which has been approved by the Bureau of Naval Personnel for use as an aid to Navy recruiters in selecting candidates for naval service. Substantial personnel attrition savings are expected through the use of this standardized actuarial system.

Related to the problems of maintaining optimal performance of Navy men on the job, NMNPRU has been conducting intensive studies in its newly equipped laboratory on the behavioral effects of fatigue, sleep deprivation, and abnormal work-rest cycles. These nationally recognized sleep studies conducted at NMNPRU were recently featured on CBS television's program "21st Century" with Walter Cronkite. In the future, such efforts are expected to assist in the development of a capability of monitoring human performance through neurophysiological and even biochemical functions.

Not limited to studying psychiatric effectiveness, human effectiveness research also reaches into the area of underwater medicine. At the Submarine Medical Research Laboratory (SMRL), Submarine Medical Center, New London, Connecticut, recent accomplishments include a new color coding system for divers and improved understanding of underwater communications problems among divers. In March 1969 one of the researchers at SMRL, Doctor Jo Ann Smith Kinney, was one of only six recipients of the Ninth Annual Federal Woman's Award for 1969. Dr. Kinney, as the leading United States expert on underwater vision problems, has conducted studies involving the selection of men with night vision suitable for nighttime naval operations, the effect of submarine service on vision, and the improvement of visibility for SCUBA divers.

Research on human isolation is being completed at the Naval Medical Research Institute (NMRI), Bethesda, Maryland, with resultant findings being applied to important field studies for selection of members and monitoring of crew effectiveness in underwater habitats. Also, new studies of behavior in hyperbaric environments are being planned.

In still another major operational area involving the behavior of man under stress, our Naval Aerospace Medical Research Laboratory (NAMRL) at the Naval Aerospace Medical Institute, Pensacola, Florida, has developed improved systems for predicting the successful performance of naval aviators. This results in a great savings to the Navy through a highly productive aviator selection program.

VACANT BILLETS

The Research Division expects to have the following vacant billets for medical officers during the summer of 1970:

One billet is for a Captain, Medical Corps as Commanding Officer, U.S. Naval Medical Research Unit No. 3, Cairo, UAR, where research is conducted on pathogenesis, epidemiology, prevention and treatment of infectious diseases. Applicants should have background and training in either research, internal medicine, public health or preventive medicine.

Two billets for Lieutenant, Medical Corps will be at the Naval Medical Field Research Laboratory, Camp Lejeune, North Carolina, where research is conducted on field and amphibious medicine. Present incumbents of billets are involved with research on etiology and prevention of acute respiratory disease of Marine Corps personnel.

Medical officers desiring consideration for assignment to these billets should submit a letter request via the chain of command to BuMed Code 71. Requests should include a current curriculum vitae.

DENTAL SECTION

PERSONNEL AND PROFESSIONAL NOTES

PARTICIPATION IN PROFESSIONAL BOARD EXAMINATIONS

BUMEDINST 1500.4D establishes the standards and procedures for participation in professional board examinations. The directive states, in part, that BUMED review of applications is no longer required and applicants may communicate directly with the examining agency for evaluation and determination of eligibility.

However, for the purpose of budgeting, at least four months prior to requesting payment of fees and travel, officers should advise BUMED, Code 6112, of their intention to take a specialty board examination.

SMILE POWER

The theme of the 22nd Anniversary of the National Children's Dental Health Week, February 1–7, 1970, sponsored by the American Dental Association, is SMILE POWER. It also marks the fourth year that the Naval Dental Corps will conduct a worldwide Preventive Dentistry Program for eligible dependent children (BUMEDINST 6610.1).

Each year dental personnel have enthusiastically participated in the program and have provided an extremely valuable service. This is commendable, but continued success of the program will once again depend on advance program planning and active participation by all hands.

All dental facilities are encouraged to publicize their Preventive Dentistry Program for two or more weeks prior to its initiation through all available news media. To promote this particular area of your overall patient education program, this Bureau has distributed 28 articles on dental health. All articles have been numbered and should you be missing articles, copies should be requested from Code 6114 of this Bureau.

Navy Preventive Toothpaste should be requested by letter to BUMED, Code 6114, not later than 5 January 1970.

CENTRALIZED PHOTODOSIMETRY FILM PROCESSING CENTER

BUMEDINST 6470.8 established the policy regarding the evaluation, including processing, of photodosimetry film. The Radiation Safety Department, National Naval Medical Center, Bethesda, Maryland, evaluates photodosimetric film on a Navy-wide basis. The directive sets forth the proper procedure for obtaining the instructions and forms required to utilize the services of the Film Processing Center.

MANUAL OF THE MEDICAL DEPARTMENT

Changes 51 and 52 to the *Manual of the Medical Department* have recently been distributed. Information of particular importance to dental personnel is as follows:

Change 51

This Change:

a. Modifies 6–109 (4) and 16–14 (3) to provide for destruction of the empty Dental Folder DD 722–1, at time of closure or termination of Health Record.

b. Changes 15–43 (4) (c) to eliminate requirement for SF 603, Dental, on Naval Academy candidates.

Change 52

This Change:

a. In 6–2 (2), 6–4, 6–6, and 6–11, reflects recent organizational modifications to the Dental Division.

b. Updates Chapter 6, Section XVI, Dental Officer Training.

Holders of the *Manual of the Medical Department* should insure the receipt and entry of these Changes.

PROFESSIONAL RELATIONS PROGRAM

PEOPLE TO PEOPLE—TAIWAN

Two dental officers and two dental technicians attached to the Dental Department, Taipei, Taiwan, recently participated in the People to People Program for Taiwan aborigines. Over 500 patients were examined. Treatment included the extraction of 744 teeth and root tips, other humanitarian treatments for the relief of pain, and instruction in oral hygiene.

Because of the poor nutrition and soft diets of the aborigines, their alveolar bone was noted to be easily compressible and the extraction of the teeth and retained roots was an extremely simple procedure which required a minimum of surgical equipment. Captain A. P. Giammusso, DC USN, is the Senior Dental Officer at Taipei.

VENEZUELAN NAVAL OFFICERS VISIT NAVAL DENTAL SCHOOL

Captain M.A. Fernandez, Venezuelan Navy dental officer, recently visited the Naval Dental School to observe the School's clinical and educational facilities. He was accompanied by Admiral J.P.T. Morales, also of the Venezuelan Navy, who is in Washington with the Inter-American Defense Board.

Doctor Fernandez, who has completed three years of study in Orthodontics at the University of St. Louis, was touring the United States prior to his returning to private practice in Caracas.

ARTICLES AND ABSTRACTS

BOND STRENGTH AT THE INTERFACE OF NEW AND OLD SPHERICAL AMALGAM

LCDR G. L. Scott, DC USN and
LCDR R. J. Grisius, DC USN.

Under some circumstances an operator may desire to repair an amalgam restoration rather than completely remove and replace it. Studies with conventional alloy have shown variations in the degree of bonding as well as the strength of the bond. However, there have been no studies on the bonding qualities of spherical alloy. The purpose of this study was to test the bonding qualities of spherical alloy when bonded to itself after varying periods of time. A metal mold was fabricated with 5 dumbbell-shaped slots .75 inch in length with a cross-sectional area of .01 inch^2. To simulate a repair, one half of the dumbbell was filled and the other half was completed after varying periods of time: 45 seconds, 15 minutes, 24 hours, 3 days, and 7 days. A total of 50 test samples were made and compared to 10 control samples. The controls were fabricated as solid specimens using the same mold. After aging for 7 days the dumbbell-shaped objects were placed in two metal holding devices, and a vertical force was gradually applied. The force required to separate the samples was measured, and this was considered to be the bond strength. Simulated repairs made after 45 seconds showed no significant difference in strength from the control samples. Significant decreases in bond strength were 30% after 15 minutes, 55% after 24 hours, 57% after 3 days, and 61% after 7 days. It was concluded that repairs may be made on fresh spherical amalgam within 15 minutes. After that time, repairs should only be made in areas of low stress or away from areas of cuspal impact.

(Abstract by Research Work Unit: MR005. 19–6052 by LCDR G. L. Scott, DC USN and LCDR R. J. Grisius, DC USN.)

THE INFLUENCE OF FILE SIZE ON MECHANICAL PREPARATION OF CURVED ROOT CANALS IN MOLAR TEETH

LCDR R. A. Murphy, DC USN and
LCDR N. H. Tracy, DC USN.

The accurate preparation of curved molar canals remains one of the most difficult and demanding aspects of root-canal therapy. A continuing problem is the selection of the proper size file for enlarging canals without ledging or perforation. The purpose of this study was to determine which size instrument will best prepare curved canals of molar teeth. The

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The opinions and assertions contained herein are those of the authors and are not to be construed as reflecting the views of the Navy Department or the naval service at large.

degree of curvature of 53 extracted intact molar teeth with curved roots was measured, and the canals were instrumented with endodontic files, sizes 10 to 50, and filled with silver cones and sealer. After 1 week, preparations were cut longitudinally and examined for ledging or perforation. It was observed that as file size increased more and more ledgings and perforations resulted. The maximum size files that accurately adapted to given degrees of curvature were determined, and it was found that the proper maximum file size for any known degree of curvature could be calculated from the following formula: file size = (110 minus angle of curvature) divided by 2. From these results it was apparent that endodontic failures in curved canals may be due to use of files too large for the particular angle of curvature and that there is no absolute file size that should be advocated for use in all curved root canals. However, since the majority of curved canals have less than 60 degrees of curvature, it was concluded that size 25 will negotiate most curved canals.

(Abstract by Research Work Unit: MR005. 19–6052 by LCDR R. A. Murphy, DC USN and LCDR N. H. Tracy, DC USN.)

PULPAL RESPONSE TO FOUR RESTORATIVE MATERIALS

S. N. Bhaskar, D. E. Cutright, J. D. Beasley, and R. C. Boyers, Oral Surg 28(1): 126–133, July 1969.

This study compares the pulpal response to four types of restorative material. The materials compared are: ZOE (Zinc Oxide and Eugenol); IRM (Intermediate restorative material — a commercial product consisting of polymethylmethacrylate- reinforced zinc oxide and eugenol); EBA (powder-Zinc oxide 57%, Aluminum oxide 28%, a copolymer of methylmethacrylate 9.5%, Rosin 5.5%, liquid-o-Ethoxybenzoic acid 66 2/3% Eugenol 33 1/3%); CAZI (powder- Zinc phosphate 85%, Zinc oxide 15%, liquid-Isobutyl-a-cyanoacrylate monomer (sterile)).

A total of seventy-eight teeth in three miniature pigs were used, and sectioned at 1, 2, & 3 weeks after placement of the materials. The materials were placed in class V cavity preps.

The pulpal response was judged according to the degree of odontoblastic disruption, inflammatory cell infiltration, and formation of new (reparative) dentine.

The results showed that odontoblastic disruption and inflammatory infiltration were least marked with the ZOE, and most prominent with the CAZI. Conversely the layer of reparative dentine formed was more prominent under the CAZI and EBA materials than under the other two.

The conclusion is that in cases where a thick layer of reparative dentine is desirable, a cavity base with EBA or CAZI is preferable. However, if the object is a semipermanent restoration, then physical properties and resistance to the oral environment should be the decisive factor.

(Abstracted by LCDR Nelson T. Crowell, DC USN.)

SCANNING ELECTRON MICROSCOPY STUDIES OF DENTAL ENAMEL

Seymour Hoffman, William S. McEwan, and Charles M. Drew.

A preliminary investigation was conducted to determine the potential capability of the *scanning electron microscope* as applied to studies of sound, untreated and experimentally treated enamel. Characteristics were noted similar to those previously demonstrated by replication technics, conventional electron microscopy and microradiology but never before observed with the *depth of field* and directness which this microscope provides. Other features were observed which added information in presently controversial areas and which have never been seen before. Gross *enamel* samples were studied, some of which were treated for varying timed intervals with *acetate buffer* pH 4.0, some with EDTA pH 7.94 and some which were not treated. The magnifications ranged from 20X to over 100,000X. Acid etched samples revealed sequential patterns of demineralization which appeared to course down the *prism cores* while EDTA treated specimens seemed to have the reverse effect, dissolution apparently occurring at *rod peripheries.* Two zones were revealed by 7-hour EDTA treated samples; a surface, "*prism-less" zone* and a subsurface zone of column-shaped structures considered to be enamel rods in various stages of dissolution. Surface coverings were also seen which appeared to have root-like extensions continuous with sub-surface structures and which were felt to be an integral part of the enamel. A hypothesis was suggested that this "*enamel skin*"

might probably represent the last-formed portion of the enamel matrix in amelogenesis, which remained unmineralized and was in direct continuity by root-like processes with the underlying mineralized matrix. The possibility was suggested that this surface coating could serve as a bed for the deposition of *extra-dental depositus* in the build-up of acquired coatings or *dental plaque*. Thus continuous organic pathways may be present on enamel surfaces for bacterial invasion and/or the diffusion of their metabolic products, from the outermost layers of infected coatings to the sub-surface zone of enamel prisms. These features were related to the formation of sub-surface lesions.

Bacteria-like structures were also seen which resembled cocci and lactobacilli forms.

It was concluded that the scanning electron microscope may be an exceptionally useful instrument for studies in dental enamel and for other basic disciplines as well.

(Abstract of work accomplished by Research Work Unit MF005. 19–6051.)

EFFECT OF FLUORIDATION ON DENTAL PRACTICE

Dent Abs 14(8): 449–450, Aug 1969.

Fluoridation, contrary to reports by other researchers, has apparently had little effect on dental practice, according to initial research finding at the University of Illinois College of Dentistry. Preliminary results indicate there is little difference between dental practice in communities with fluoridated water and communities whose water has a fluoride deficiency, according to Bruce L. Douglas, principal investigator for the research project which has compared dental practice in eight sets of communities. Douglas, professor of community dentistry, Miss Sylvia Coppersmith, field director, and a laboratory program administrative assistant, began the project with the hypothesis that fluoridation alters the nature of dental practice. They were assisted by Donald A. Wallace, professor of dental radiology, and a *Dental Abstracts* consultant. They expected to find variations in dental treatment, patient loads, dentists' incomes and practices in communities with fluoridated water. However, they found an apparently similar number of patients who seek dental treatment in both types of communities. They also found that most patients apparently visit a dentist when they are in pain or obviously in need of care; few people go for preventive dental care.

One possible explanation for fluoridation's apparent minimal impact on dental practice is that population growth has outstripped the availability of dentists to attend to the dental needs of the public, even in fluoridated communities.

The investigators also found that fluoridated water has little effect on the age at which children first visit a dentist's office—also contrary to original expectations.

Communities that were studied are: Aurora and Freeport-Kankakee, Ill; Kewanee and Centralia, Ill; Marion and Sandusky, Ohio; Joliet, Ill, and Mansfield, Ohio; Elwood and Connersville, Ind; Huntington and Shelbyville, Ind; Frankfort and Crawfordsville, Ind; Pueblo, Colo, and Beaumont, Texas. (The first city in each pair has water that is optimally naturally fluoridated.)

NURSE CORPS SECTION

The following paper by LCDR Nancy L. Lundquist, NC USN was written to provide guidelines for Nurse Corps officers who are working in nonpsychiatric areas where behavioral problems exhibited by patients hinder effective patient care. A portion of the paper is being utilized by the American Red Cross in counseling mothers of problem children. LCDR Lundquist is an instructor assigned to the Neuropsychiatric Technician School at Philadephia Naval Hospital.

DISCIPLINE OF PATIENTS—A NURSING RESPONSIBILITY?

Nurses are often confronted by patients who disrupt ward routines in order to satisfy their immediate individual desires. Many such patients display behavior patterns which will respond to discipline, yet

nurses are often unwilling or unable to assume a role in the disciplinary process. Recognition of troublesome behavior patterns, their underlying causes and resulting effects on involved persons, leads to the realization that discipline has to be a nursing responsibility. Once this concept is accepted, then the nurse can focus on the process of effecting discipline.

Everyone responds to stress emotionally and physically. Hospitalization by its very nature presents many stressful situations which particularly threaten the self-image. Anxiety is experienced. Since this internal sense of tension is uncomfortable, most people will try to rid themselves of it. In an attempt to do so, seemingly well adjusted individuals may revert to immature methods of seeking a more comfortable state. Under stress, we regress. Individuals already predisposed to react unfavorably to stress may attempt to "act out" their inner tensions. Individual behavior varies, as does individual concept of what is self-threatening. To one it may be a disfiguring injury; to another it may be a lengthy and incapacitating illness; and, to still others it may be hospitalization itself. It is evident that many patients will resort to disruptive behavior in order to relieve themselves of anxiety.

Obvious disruptive behavior such as boisterousness, vulgarity, getting into fights or racing in wheelchairs, is quickly detected. Less conspicuous and more troublesome is the patient who acts out in subtle ways. He procrastinates, disagrees with routine, pouts or "forgets" to keep appointments. On the other hand, he can be the most charming patient on the ward. His charm, however, is a manipulative tool and remains only so long as he succeeds in getting what he desires. For example, consider this patient. Amazingly, one doctor signs multiple special liberty requests for him, the corpsman wakes him up last at reveille, and a nurse permits him to watch TV past 2200. However, when he becomes bored or when certain personnel do not adjust to his style of living simply because it deviates from the accepted ward routines, he reacts explosively and angrily.

One might argue that it would be easier to bend the rules somewhat, that patients would be less likely to cause trouble if a more permissive attitude prevailed. The time spent in dealing with disruptive behavior represents nursing care time lost to other patients on the ward. Since "acting out" patients do not assume their fair share of ward work and responsibilities, a heavier share of the work is assumed by the remaining patients. After having had their care disrupted or sleep disturbed by the behavior of "acting out" patients, others begin to wonder why such behavior goes unchecked. They begin to question the competence and leadership of the nursing staff: "If I came back from surgery and were totally dependent on their judgment, would I be safe in their hands?" When patients start asking questions of this nature, it is certain that their anxiety level will increase and ultimately inhibit their clinical progress.

When disruptive behavior negatively affects patients on the ward restoration of control becomes a nursing responsibility. How can this responsibility be met? First, the staff members must meet in order to clearly define the problem behavior. Having done this, the staff must "brainstorm" and outline limits that will effectively curtail that particular behavior. For instance, "grounding" an orthopedic patient by taking his wheelchair away for an hour or two will probably convince him that racing around the ward is not acceptable behavior. However, if he continues to "act out", his grounding time can be increased until he is persuaded to change his behavior. Limit-setting is a process which ultimately educates the patient concerning what he can and what he cannot do. It is not, however, rigid control of all behavior; it is planned and consistent curtailment of problem behavior only.

The key factor in enforcing limits is consistency. One dissenting staff member can ruin even the best plan for discipline. It is, therefore, essential that the charge nurse persuade all staff members, and particularly the WMO, that their support is needed. It is often helpful to point out that discipline is more of a caring rather than a punitive process. Remind staff members that, for the child, the first denials in the home have to do with protection. Parents who care about their children set limits on behavior that could ultimately be harmful to them. We owe the same kind of concern to our patients. It is tempting, for instance, to bend the rules in order to provide immediate gratification for a young amputee. But what are we really giving him? Certainly not the indicated preparation for facing the ultimate reality of the nonhospital world.

Sometimes the disruptive behavior persists, in spite of a goal-directed plan of care. Usually the problem is related to inconsistent support by one of the staff members despite persuasive efforts by the charge nurse. Occasionally a doctor may be quite permissive and frustrate attempts to set reasonable limits on patient behavior. It then becomes the charge nurse's duty to discuss the problem, with her immediate supervisor if necessary, so that it may be

pursued at a higher level for appropriate action. The dissenting staff member can appear at any level. It may be, for instance, a young nurse with a strong need to be liked and an inordinate fear of failing to please even the problem patient. On the other hand, it may be a corpsman who satisfies a personal need to flout authority by not supporting the decision to set limits. Rivalries between staff members are often discovered when it becomes apparent that one individual is resisting a policy merely because another supports it. These staff members need to be counseled on an individual basis as to the motivation for their nonsupport. They should be acquainted with the expectations of the charge nurse concerning their behavior. If the nursing staff member fails to cooperate despite counseling, then the problem should be resolved administratively.

It is clear that nurses can anticipate disruptive behavior in the hospital setting and that this behavior will negatively affect all patients involved. Curtailing such behavior by a disciplinary process called setting limits, is a nursing responsibility. The persuasive skills of the charge nurse are vitally important in securing staff cooperation in support of the limits decided upon. Consistency is the key to effective discipline.

OCCUPATIONAL MEDICINE SECTION

INFLUENZA IMMUNIZATION AND LOST TIME IN INDUSTRY

J Occup Med 11(6): 311–317, June 1969

The prevention of catastrophic or epidemic disease through active immunization of a susceptible population is a logical answer to the problem of a growing population faced with a shortage or maldistribution of physicians and ancillary personnel.

The ideal immunizing agent should have the following characteristics: (1) the vaccine should invariably produce effective and permanent immunity with a single dose; (2) there should be complete freedom from harmful reactions of immediate, local, or systemic nature, and no delayed reactions should occur; (3) high resistance to acquisition of microorganisms should follow its use, resulting in the prevention of the carrier state among those immunized; (4) it should be administered in such a fashion as to be well accepted by the patients; and (5) microorganisms should not be shed by the vaccine recipient in such a fashion as to endanger household or community contacts, regardless of their state of health.

From these standards it is apparent that no ideal vaccines are available for use as yet, although the poliomyelitis, measles, and tetanus vaccines are better qualified than most. All vaccines fail in at least one, and many in several, areas.

Administration of influenza vaccines in industry is not justified unless it can be demonstrated that there are clear medical indications and substantial medical benefits to be derived. It must also be demonstrated that the expense involved is justifiable in the form of lessened sick absenteeism. Influenza vaccination programs cannot be justified merely as a labor relations measure, nor contrived to indicate paternal interest in the health and welfare of the worker. It falls within the domain of the industrial physician to inform management of his best judgment in these matters, since it will be in his offices that such programs will be justified or condemned.

Influenza and Mortality

The World Health Organization (WHO), the U.S. Armed Forces Epidemiological Board's Commission on Influenza, and the National Communicable Disease Center (CDC) keep watch over the disease and its viruses, and also over the influenza vaccines developed to prevent it.

It has been noted that mortality from influenza is commonly limited to the elderly, especially to the elderly with chronic disease. The course of an influenza epidemic is charted by recording the excess mortality associated with the outbreak. There are 112 large U.S. cities which collaborate with the CDC in providing a weekly count of influenza-related deaths.

Mortality reports are used, because of the notorious unreliability of official morbidity reports, as indices of prevalence of the disease. Attention is concentrated on deaths attributed to influenza and pneumonia, since reporting does not accurately distinguish between the two. Of the deaths that occur during an influenza epidemic, many of those above the normally anticipated number are ascribed to cardiovascular, renal, and metabolic disease rather than to influenza, suggesting that mortality from influenza is chiefly associated with underlying illnesses.

Diagnosis of Influenza

In the absence of epidemic conditions the diagnosis of influenza is unreliable unless confirmed by laboratory studies. In the presence of an epidemic, the occurrence of an acute febrile respiratory illness with pronounced constitutional manifestations early in its course makes the diagnosis fairly accurate, since no other disease affects so many people of all age groups over such wide geographic areas in such a short period of time. The diagnoses are even more reliable if representative cases from the area undergo laboratory confirmation.

Even in an epidemic, similar illnesses due to other causes occur. Specific etiologic diagnosis requires either isolation and identification of the virus from nasopharyngeal washings, throat garglings or throat swabs, or antibody studies of acute and convalescent serums such as complement fixing (CF) or hemagglutination inhibition (HI) titers.

Influenza Vaccines

There are, in general, two types of vaccine, a type-specific monovalent vaccine prepared with a specific virus, and a polyvalent vaccine that contains multiple antigenic strains that have caused epidemic disease in the past.

In an epidemic with a particular variant running riot, there may not be a type-specific vaccine available, and existent vaccine may be totally ineffective.

Recent Studies in Industry

Recently there have been two very significant studies evaluating the use of influenza vaccine in industry. While they are concerned primarily with the annual use of polyvalent vaccines in non-epidemic years, they each illustrate dramatically some basic truths about study design and proper conduct of epidemiological investigations under industrial conditions that would apply to many previous studies.

In the first of these studies, polyvalent vaccine was offered to all employees in a non-epidemic year. Half the volunteers were inoculated and half the volunteers were not inoculated to serve as the control group. None of the non-volunteers were inoculated.

The results were surprising. There were no statistically significant differences between the inoculated volunteers and the non-inoculated volunteers in any of the subsequently accumulated data relating to sick absences. Any effect produced by the influenza vaccination was not detectable, either with respect to frequency of absence, length of absence, number of absences per person, or type of diagnosis assigned by the private physician.

There were, however, statistically valid differences between the experiences of the volunteers and the non-volunteers. Almost twice as many non-volunteers were absent from work because of respiratory illnesses, and the rate of absence was twice that of the volunteers.

If this study had utilized only the non-volunteers for a control group, as is frequently done, one would have to conclude that the influenza inoculation had produced a 50% drop in absences in the inoculated group when, in fact, the inoculations had no detectable effect.

In the second study, a vaccination program was conducted in a large industrial population of over 10,000 for a period of 3 years. The study population was only part of the total population of the parent company and was limited to 2 locations. The vaccine was a polyvalent type and the study was conducted during non-epidemic years.

In this project only physician-certified absences of over seven days were counted as valid absences so far as the study was concerned. Absences of less than this duration in many cases involved no physician contact, and self-diagnosis was considered to be totally unreliable.

In the fall of 1961, 6,100 employees in Montreal and 5,800 employees in Toronto received the initial injection. A second dose was given in the spring of 1962, a booster in the fall of 1962, and another booster in the fall of 1963. During this time the volunteer group had become smaller with each inoculation, and by the end of the study had dropped from 11,900 to 5,500 or about 54% reduction in participation. This decline in enthusiasm has been commented on in other reports.

When the figures were analyzed, there did seem to be a significant difference between the respiratory

illness experience of the inoculated and the uninoculated groups. However, it was decided to test the validity of the results by reviewing the respiratory disease experience of the inoculated group in prior years. It was found that the excellent experience that this group had, in 1962 and 1963, was not the result of the immunizations, but was the same type of experience that the group had been having for at least 3 years before the vaccine program was initiated.

It was concluded that the vaccine had been offered on a voluntary basis to a group of industrial employees who apparently did not benefit from it, and that their respiratory disease experience was markedly better than that of the group who had not participated in the immunization program. This conclusion was further substantiated when the entire company's respiratory disease picture for comparable years was studied. The over-all company case rates for disease of the respiratory system had remained fairly constant for a 5-year period, and was not influenced favorably by the immunization program.

Apparently whatever makes a volunteer a volunteer also inbues him with more resistance, less susceptibility, or better motivation. The latter seems more likely. You wonder what deters the non-volunteer. Is it perverseness, paranoia, independence, or skepticism? In any event, the self-selection allowed in many studies when volunteer groups are compared with non-volunteer groups does not permit true random sampling of the population, and invites misleading conclusions. The factors that distinguish the volunteer and the non-volunteer probably also influences the frequency, duration, and degree of disability related to sick absence in general.

Reactions to Influenza Vaccines

As mentioned before, it makes no practical difference to industry whether an individual worker is absent from work because of an infection or because of a reaction to a vaccine intended to protect him from the infection. The end result is the same, the loss of the employee's services.

Commercial influenza vaccines manufactured in the United States are prepared in chick embryos and penicillin is not used in the process; therefore, allergy to penicillin is of no concern but egg allergies may be very important. Care must be exercised in administering the vaccine to persons who have any allergic history. Persons so sensitive that they cannot eat eggs or chicken, or with a history of allergic reaction to previous inoculation of vaccines prepared in eggs, are prone to serious allergic reactions.

In addition to allergic reactions to eggs, there are also local and constitutional reactions to the vaccine itself. Half or more of the individuals receiving injections have experienced mild local tissue reactions.

Constitutional reactions often resemble a brief and mild attack of the disease, and consist chiefly of fever, headache, malaise, nausea, vomiting, chills, and muscle aching. Only those manifestations having onset within 24 hours after vaccination could be definitely attributed to the vaccine. There is considerable variation in frequency and severity of reactions from different products (different manufacturers, different lots, different types of vaccine).

Constitutional reactions have been reported in different studies ranging in frequency from 1% to 2%, to 10%, and to as high as 25% in studies using oil-adjuvant polyvalent vaccines.

As in other studies comparing frequency and severity among men and women employees, the rate of occurrence and severity of reactions reported were greater for women than for men.

There is also a tendency in industrial immunization programs to attribute all ills that occur within 2 weeks of vaccination to the vaccination.

Workmen's Compensation

Medicolegal aspects must be considered when contemplating an industrial vaccination program. Postvaccinal reactions are held to be compensable under the workmen's compensation laws of various states.

In cases that have been heard, the employer has been considered to be less than altruistic in his interest in vaccination programs since he intends to profit by lessened absenteeism and improved employee relations. This attitude has been enough to establish a work-related status for such programs. Even when an employer has had a worker inoculated to satisfy the requirements of a state statute, there is serious doubt as to whether the employer can remain free of responsibility in the event of a reaction.

Many organizations request the employees to sign a waiver, stating that he fully understands and assumes the risks involved, and releases the company from any legal obligation in the event of untoward results. Some feel that such legal maneuverings only serve to sharpen aggressiveness and increase suspicion of the employer's motives. In any event, and regardless of the motivation of the employer, in many jurisdictions the working man is not allowed to

waive his rights under workmen's compensation statutes.

Recommendations for Vaccination

Yearly vaccination is recommended for certain high risk groups on the basis of:

Age: Individuals over 45, especially those over 65
Pregnancy
Presence of chronic debilitating diseases:
- Cardiovascular:
  - rheumatic heart disease, especially with mitral stenosis
  - arteriosclerotic heart disease
  - hypertension
  - frank or incipient cardiac insufficiency
- Bronchopulmonary disease:
  - chronic asthma
  - chronic bronchitis
  - bronchiectasis
  - pulmonary fibrosis
  - pulmonary emphysema
  - pulmonary tuberculosis
- Endocrine disorders:
  - diabetes mellitus
  - Addison's disease

These recommendations encompass the breadth of the current vaccination recommendations of the watchdogs of our domestic public health, the Surgeon General of the United States, and the CDC of the Public Health Service.

Conclusions

There is still not available a standardized, effective, influenza vaccine. Public Health authorities recommend use of available vaccine in limited segments of the population: the aged, the chronically ill, and pregnant women. The oft-quoted effectiveness rates of up to 75% appear to be based on military experiences, which are not applicable to civilian or industrial population. Even if these rates were verified, the present vaccines would not compare favorably with protective vaccines against other, more life-threatening, viral or bacterial diseases.

Studies in industry, both in epidemic and nonepidemic years, have not been able to demonstrate a high enough rate of effectiveness in the form of decreased lost time from work to justify the expense of these undertakings. There appears to be no justification of continued influenza immunization of industrial populations.

HIRING EMPLOYEES WITH NERVE DEAFNESS

J Occup Med 11(6): 319–321, June 1969.

For many years research in occupational hearing loss has concerned itself chiefly with determining how much and what kind of noise damages hearing, and in finding a valid pre-employment susceptibility test to noise. In spite of extensive efforts we still do not have definite information about these problems based on reliable data. While these investigations continue it seems proper for us to center our attention upon other practical problems confronting industry. In view of our great concern for the well-being of industrial employees, and to some degree our interest in the medicolegal aspects of occupational hearing loss, we should consider the important practical issue: "Should We Hire Individuals with Nerve Deafness for Noisy Occupations?"

Let us first explore the scope of the problem. What is the incidence of sensorineural deafness (or as we shall call it in this discussion, "nerve" deafness) among job applicants? In the paper making industry, for example, we have shown that, of over 400 consecutive job applicants (most of them under the age of 30) 2% had compensable hearing loss (over 15 dB average loss at 500, 1,000 and 2,000 cps), and an additional 2% almost fit this category. Three percent of the subjects with negative noise histories had losses in the compensable range. About 22% of the job applicants had hearing losses of 10 dB or greater in at least 2 frequencies. Fox also has shown that 27 out of 100 newly hired workers had hearing losses.

The incidence of nerve deafness among employees and applicants for employment by the Telephone System in several geographical areas is quite similar. Of 1,048 cases tested in one area, 75% reported exposure to either gunfire or industrial noise or both. Thirty-two percent with this history showed high frequency losses. In another area, 53% of 999 cases reported exposure to either gunfire or industrial noise or both. Forty percent with this history showed high frequency loss at 4,000 and 6,000 cycles.

The mean losses were 30 to 40 dB at 4,000 and 6,000 cps for men up to 39 years of age. The chief cause of hearing losses among most of these 2,047 job applicants and employees was, in all likelihood, gunfire, but many individuals had high frequency hearing losses from other causes.

If we add to these statistics the known data about hearing loss in certain noisy jobs, we must conclude that the incidence of nerve deafness in the population seeking industrial jobs is greater than is generally appreciated. Among experienced paper-machine operators, for example, it appears to be well over 75%. Among chippers, drillers, and drop forge operators it is no doubt even higher.

It is essential, then, for all industries to do pre-employment or pre-placement hearing tests in order to decide if an applicant should be hired, and to establish his baseline hearing level prior to employment. The threat of creating a new, large group of hearing-handicapped, non-hirable employees, especially veterans with gunfire exposure, is a serious problem. American industry should not be compelled to discriminate against such citizens, but rather encouraged to continue helping them find suitable jobs.

Background Information

After many years of extensive research to relate temporary threshold shift (TTS) and permanent threshold shift (PTS), we find that there are still no valid and reliable reports in which the PTS and TTS tests were done on the same individuals over a period of many years. It seems that practically all studies relating TTS and PTS are based on TTS data obtained from normal hearing subjects in a laboratory, and then compared to PTS data obtained from employees actually exposed to presumably similar noise levels.

An important problem more directly related to the topic of this paper is the susceptibility of the ear, already nerve deaf, to noise. There is no valid evidence to show that employees with nerve deafness are more susceptible to noise than are those with normal hearing. On the contrary, it may be that, within certain limits, they are even less susceptible than people with normal hearing, especially if one believes that if you do not get any TTS, then you do not get any PTS. Some of us have demonstrated rather conclusively, that ears with permanent hearing loss are likely to show less TTS than do normal ears.

Now possibly we can answer with more clarity the question: "Should we hire applicants with nerve deafness?" Since individuals with nerve deafness have not been shown to be more sensitive to further noise damage, we may feel free to hire such people if we bear the following concepts in mind:

1. We should be able to assure ourselves and the applicant that if he uses adequate ear protection provided by the company, his hearing will not sustain any substantial additional damage due to the environmental noise at his job.

2. Legislation and regulations, local, state, and federal, should protect the employer from liability for hearing loss that his company did not cause.

3. The applicant's hearing level should meet the demands of his job, and he should not create a safety hazard as a result of impaired hearing, or as a result of excessive attenuation of hearing with ear protectors.

4. We must observe the proviso that the applicant's hearing loss should not be a progressive nerve deafness.

RESPIRATORY TRACT IRRITATION FROM FIBROUS GLASS INHALATION

J Occup Med 11(8): 409–410, August 1969.

During the past several years, there have been a number of reports published which discuss the effects of fibrous glass upon the lungs. The authors of these reports have been primarily concerned with the possibility that inhalation of finely divided particles of fibrous glass may produce fibrotic pulmonary disease. The weight of evidence derived from these investigations seems to indicate that neither in the experimental animal nor in the occupationally-exposed worker is inhalation of fibrous glass particles likely to produce permanent lung changes.

Having no relevant information, the authors cannot comment upon the relationship between fibrous glass inhalation and pulmonary fibrosis or permanent pulmonary damage of any kind. Attention should be focused, however, on one health aspect of fibrous glass exposure which, although well-known among fibrous glass workers, has not been reported elsewhere, apparently having been obscured by scientific concern over more serious pulmonary disease. This problem is respiratory tract irritation which is sufficiently serious to compel the worker to seek medical attention, and is characterized by one or more of the following: bronchitis, rhinitis, sinusitis, pharyngitis, and/or laryngitis.

In California, during the period extending from January 1960 to June 1962, 691 cases of occupational disease were attributed to fibrous glass exposure. Of these, 38 were primarily problems of respiratory tract irritation; the remainder involved the effects of fibrous glass on the skin and eyes. These cases were summarized in an internal health department document, and the original reports discarded.

During the interval between June 1962 and September 1967, no special summarization of reports of occupational disease attributed to fibrous glass exposure was compiled. The original reports received during this period have also been discarded. Remaining available for study are reports received from September 1967 through September 1968. During this 13-month period, 28 reports describing adverse respiratory reactions to fibrous glass were received. In all, then, 66 reports of respiratory tract irritation attributed to inhalation of fibrous glass were received during these two periods.

Among the 66 cases were 11 females and 55 males, ranging in age 17 to 68 years. Occupational designations indicated that cases occurred primarily in individuals who worked directly with fibrous glass or fibrous-glass-bearing plastics, especially where a dusty operation such as cutting, sanding, or machining was involved. In addition, however, a number of case reports described individuals who merely worked in the general area where fibrous-glass-containing dust was being generated. Thus, it appears that working with the material is by no means the sole criterion of exposure.

On 19 of the 28 case reports received during the period of September 1967 through September 1968, a notation was included which indicated that a chest roentgenogram had been taken. This information on the 1960–1962 reports is not available. Of these 19 reports, 13 were accompanied by some statement of roentgenographic evaluation. These 13 included 9 negative reports, 1 "bronchitis," and 1 "pulmonary fibroisis." The physician who reported this latter case expressed some doubt that the observed "pulmonary fibrosis" was related to the patient's fibrous glass exposure.

Three of the 66 cases resulted from the same exposure, and were reported by a practicing occupational medicine specialist.

Case Reports

Case 1. This 48-year-old male maintenance employee worked several 8-hour shifts, removing fibrous glass insulation from around a steam pipe.

He wore no respiratory protective device, even though such equipment was available. His chief complaints were sore throat, congested nasal cavities, pain in the larynx, and skin irritation.

Physical examination revealed no ear, nose, or throat abnormalities, and the skin was clear. The chest x-ray was reported as normal. The physician's impression was that there were minor irritations of the respiratory tract from fibrous glass dust. When the employee began to use the respiratory protective device provided, the respiratory irritation lessened, but the itching and irritation of the skin continued.

Case 2. This 27-year-old male maintenance employee worked several 8-hour shifts without respiratory protection, removing fibrous glass insulation. His chief complaints were a cough and an inability to sleep because of itching.

The physician's examination revealed a slight injection of the pharynx. In addition, the nasal mucosa was red and crusted. There were slight rhonchi in the lungs, which cleared on coughing. A chest x-ray was reported as having prominent markings in the left lower lobe which were probably vascular and within the limits of normal. The employee's symptoms abated after he began wearing a respirator.

Case 3. This 34-year-old male supervisor was in the area without respiratory protection, directing the removal of fibrous glass insulation, for three 8-hour shifts. When he came to the dispensary, his chief complaints were persistent throat irritation, cough, and a burning sensation in the throat. He reported a long history of sinusitis, allergic rhinitis, and asthmatic episodes occurring in the past as a result of ingestion of chocolates and other foods. There was no asthma attack with this work exposure.

The physician's examination revealed some rhonchi in the left lower lung field. A chest x-ray was interpreted as normal. The symptoms were gone the following day.

Conclusions

Although these reactions to fibrous glass do not, to our knowledge, result in permanent disability, the authors believe such reactions are nonetheless noteworthy because they indicate that fibrous glass is a substance which is fully capable of causing symptomatic respiratory irritation. Therefore, the following recommendations would seem to be in order:

1. Workers should be protected from respiratory exposure to airborne fibrous glass by accepted industrial hygiene practices. Reliance on filter-type respirators should be confined to brief exposure situations.

2. Further studies should be undertaken to define more clearly the health hazards associated with exposure to airborne, fibrous glass-plastic mixtures.

WELDERS' SIDEROSIS

Arch Environ Health 19(1): 70–73, July 1969.

Exposure to iron oxide dust or fumes occurs in a number of occupations including arc welding, silver polishing, iron grinding, and mining and milling of hematite and magnetite ores. Sustained inhalation of significant concentrations of iron oxide can produce roentgenographic changes characterized by reticulation with micro or miliary nodulation over both lung fields. Doig and McLaughlin have reported the cases of two welders in whom the nodular x-ray densities disappeared in one case after removal from exposures to iron oxide, and diminished in the other, when the exposure was reduced. There is some controversy as to whether chronic inhalation of pure iron oxide dust or fumes produces solely asymptomatic or benign pneumoconiosis. Some of the more recent literature has indicated that chronic exposure to iron oxide dust can produce symptomatic pneumoconiosis. From a statistical and clinical study of cases of pneumoconiosis encountered among the iron workers of the Briey Basin in France, Casanouve and Soudan concluded that siderosis occurs frequently and is characterized by respiratory symptoms and reticulonodular shadowings of the chest roentgenograms. These authors also pointed out that longstanding siderosis can lead to cor pulmonale. More recently, Stanescu et al have reported on the pulmonary mechanics in arc welders' siderosis and found that seven out of 16 welders studied had some exertional dyspnea and 3 had a chronic cough. These symptoms did not correlate well with smoking. Although the spirographic values were generally within the normal range, the arc welders had a statistically significant reduction in static and functional compliance. Seven had a functional compliance below the lower limit of the control group of 13 healthy, unexposed men. The authors postulated that the decrease in pulmonary compliance in arc welders could be explained by iron deposits per se or possibly an associated fibrosis, or both. They also suggested the possibility that agents other than iron oxide fume and dust, such as ozone, nitrous oxides, and substances present in welding fumes from rod coatings, may contribute to the pulmonary damage.

Recently, we conducted a clinical, roentgenographic, physiologic, and environmental study of a group of welders, and the data obtained in this study serve as a basis for this report.

Methods

Twenty-five welders comprised the study group. Each individual underwent a detailed medical, social, and occupational history, a clinical examination, a chest roentgenogram, and a battery of pulmonary function tests. The pulmonary function tests included forced expiratory volume (FEV), one-second forced expiratory volume (FEV_1), residual volume (RV), total lung capacity (TLC), and pulmonary diffusion capacity for carbon monoxide (DL_{co}). The FEV and FEV_1 were measured by a Krogh spirometer and the volume changes of the spirometer were electrically recorded on a strip chart recorder. Residual volume was determined by a nitrogen dilution method using the closed circuit technique as recommended by Wright and Gilford. All lung measurements were determined at ambient temperature and pressure, saturated (ATPS). The values for RV were corrected for the dead space of the breathing tube and the phase of expiration in which the subject was connected to the spirometer. The DL_{co} was determined at rest by a single breath technique described by Ogilvie et al. Predicted values for FEV, RV, TLC, and ratio of RV to TLC were calculated on the basis of the best regression equations formulated by Needham et al. For DL_{co}, the percentage predicted was based on the equation of Ogilvie et al:

DL = Surface Area (in square meters) x 1,885–6.8

The control group was comprised of 20 men of similar age distribution who resided in the same area, but who had no occupational dust exposure. The 95% confidence intervals for all the lung parameters were based on the data obtained from the control group.

Results

Clinical—The welders ranged in age from 25–70 years with a mean of 48.8 years. Age of the controls ranged from 25 to 64 with a mean of 46.7 years. The difference in the mean ages of the two groups was not statistically significant.

Two of the welders had a chronic cough, one of 4.8 years' and the other of 5.4 years' duration. In the former, the cough was productive. Two of the controls had a chronic cough of 2.7 and 4.2 years, respectively, and in 1 the cough was productive. None of the welders or controls had dyspnea. Lung crepitations were present in 2 welders and rhonchi in 1. None of the controls had abnormal lung findings. The prevalence of cough and abnormal lung findings in the welders and controls was not significantly different.

A postive smoking history was obtained from 14 of the welders and 11 of the controls. The degree of smoking (number of cigarettes smoked multiplied by the duration of smoking) between the 2 groups was not significantly different. The criterion for a positive history was smoking 20 cigarettes a day for a minimum of 5 years.

Roentgenographic—Eight of the welders had roentgenographic findings characterized by reticulonodular shadowing in both lung fields. The controls had normal chest roentgenograms.

Physiologic — The mean values of the pulmonary function parameters in the welders and controls fell within normal range, and the mean values between the two groups were not significantly different.

Environmental—The plant facilities studied are used for the manufacture of welded, sheet metal products such as cesspools, tanks, equipment for water treatment, and laundering machinery. Both stainless steel and black iron ore are used in the fabrication of these products. Air samples taken during this study (1968) were collected from inside the welders' face shields and from points close to the external surface of the shield. General air samples were taken, and air sampling was also done at a flame cutting operation. Chemical tests for ozone were performed upon samples obtained at shielded arc welding operations, and also for fluorides, since a fluoride flux rod was used in one of the welding processes. Concentrations of iron oxide inside the protective face shield ranged from 0.65 mg/cu m to 1.7 mg/cu m and from 1.6 to 12.0 mg/cu m outside the shield. Iron oxide concentration in general air was below detectable levels. The concentration in the breathing zone of the worker engaged in flame cutting was found to be 3.0 mg/cu m. The flame cutter wore no face shield at the time the air sampling was done. Concentrations of ozone were found to be negligible, but 2.7 mg of fluoride was found in the air sample collected outside the face shield of the worker using the fluoride flux rod in welding.

Previous samplings for iron oxide taken in this plant in 1960 and 1961 revealed iron oxide concentrations ranging from 30 mg/cu m to 47 mg/cu m at the breathing level of the welders. The plant made improvements in the ventilating system shortly after 1961.

The duration of exposure for the overall group of welders in the present study ranged from 3 to 32 years with an average exposure of 18.7 years. Twelve of the welders had an exposure of less than 20 years and 13 had that of 20 or more years. Four welders had less than 8 years of exposure.

Comment

Clinical and Environmental—The present data indicate that sustained exposure to iron oxide (average, 18.7 years) did not produce clinical or physiologic findings in the welders which were significantly different from those of the control group. There was a moderate prevalence of positive roentgenographic findings consistent with siderosis in the overall group of welders, with a greater percentage of these findings in those exposed over 20 years. The air concentrations of iron oxide determined during the present study are below the threshold limit value of 10 mg/cu m as recommended by the American Conference of Governmental Industrial Hygienists (ACGIH). However, the present levels do not completely represent the degree of exposure of the workers during their entire employment, since previous tests in 1960 and 1961 revealed concentrations of iron oxide markedly in excess of the present threshold limit values. Most of the workers examined worked in the plant prior to 1960. Improvements in the plant ventilating system, therefore, account for the lower levels of iron oxide found during this study, and the finding of siderosis in 8 of the 25 welders is indicative of their prolonged exposure to the higher concentrations found prior to 1961.

Correlation of Clinical, Roentgenographic, and Physiologic Findings With Duration of Exposure — In general, there was no correlation between the clinical, roentgenographic and pulmonary findings, and duration of exposure. The incidence of chronic cough, positive findings in the chest, and abnormal chest roentgenograms in those welders exposed to iron oxide dust for less than 20 years was not significantly different from those exposed 20 years or more. Similarly, there was no significant difference in the mean values of FEV, FEV_1, and DL_{co} of those exposed less than 20 years and those exposed 20 years or more. In general, the data demonstrate that prolonged exposure to iron oxide among welders in this study has not produced significant clinical and physiologic alterations when compared to the control group.

FROM THE SURGEON GENERAL

The Secretary of Defense has again expressed his interest and concern in current programs designed to assist men separating from military service. He has asked for a review of separation procedures, including support of special activities such as TRANSITION, at all military installations in the continental United States and overseas. The purpose of the review is "to insure that we are providing vigorous, uniform programs for all separating personnel which will help them toward new goals as they reenter civilian life." Although the Bureau has not been asked to participate directly in the review, I welcome this opportunity to discuss separation counseling with you.

Some personnel are leaving the service without the counseling required under TRANSITION or that provided by the Veterans Administration and other Federal agencies. Included are men with serious disabilities received in Vietnam who can be expected to have major problems of adjustment upon return to the civilian community. We do not know how many servicemen have gone home without proper counseling. Unfortunately, some of these counseling failures have been aired on national television.

I fully appreciate the burdens being placed upon Navy medical facilities and that the shortage of personnel does not permit providing full-time personnel to each new task such as TRANSITION. It is not my intent to criticize efforts in this area. Rather I would ask that you reexamine accomplishments in the light of goals set by current directives; if improvements can be made, they should be made expeditiously. I would ask further that you give this matter your personal attention so that the Medical Department's contribution is adequate to the intended goal.

Captain N. B. Curtis, MSC USN, Director, Patient Affairs Division, telephone extension 61236, is coordinating separation counseling material.

MARINE CORPS MEDICAL SUPPORT DESK ESTABLISHED IN BUMED

In order to consolidate responsibility for the various aspects of BUMED support to the U.S. Marine Corps, a new Bureau Division has been established by the Surgeon General under the Assistant Chief of Bureau for Research and Military Medical Specialties. The Division (Code 75) has been named "Marine Corps Medical Support Division" and CAPT J. H. Stover, Jr., MC USN, has been assigned as Director. CAPT Stover's last assignment was Force Surgeon, Third Marine Amphibious Force and his previous assignment was as Commanding Officer, Naval Medical School, National Naval Medical Center, Bethesda, Maryland.

It is planned that the role of the new Division will be to administer and coordinate all BUMED matters pertaining to the Marine Corps including personnel assignments,organization, logistics, research and development requirements and training. The Director is assignments, organization, logistics, research and development and Educational Center, Quantico, Virginia and maintains close liaison with the Staff Medical Officer, Headquarters, USMC (CAPT R. Witwer, MC USN).

RORER AWARD PRESENTATION

The Gastroenterology Clinic and Research Branch of the Naval Hospital, Bethesda, Maryland, has just received word that an original clinical study, "Gastric Analysis in the Evaluation of Post-vagotomy Gastric Stasis" won the second prize of $300 in the 1969 William H. Rorer, Inc. contest for the best unpublished paper. This paper was presented at the 34th Annual Convention of the American College of Gastroenterology in Houston, Texas on Wednesday morning, 22 October 1969. The paper will be published in the American Journal of Gastroenterology. Sharing the award are CAPT William M. Lukash, MC USN, LCDR Raymond B. Johnson, MC USN, and LCDR J. Raymond Fletcher, MC USN.

TRANSFER TO VA HOSPITALS

BUMEDINST 6320.11B provides guidance in the policies and procedures for transfer of active duty personnel to Veterans Administration hospitals. The Surgeon General has previously requested that certain patients be transferred to the VA hospital system as early as their medical condition will permit. Particularly involved is that category of patient who is readily identifiable for future separation or retirement by reason of physical disability and who will require immediate further hospitalization in the VA hospital system after retirement or separation.

Personnel processing procedures should promote transfer of such patients to a VA hospital to begin rehabilitation as soon as their clinical status permits the move. Hospital admissions with conditions such as severe injury to the nervous system (including

quadriplegics, hemiplegics, and paraplegics), blindness and deafness requiring definitive rehabilitation, major amputees, and other diseases or injuries which obviously preclude return to duty, should be expeditiously prepared for transfer to a VA hospital. Medical records and statement of service should be promptly requested for use by the PEB. Request to ASMRO for a bed designation should be submitted as soon as a medical board is held and further treatment in a VA hospital is recommended. Medical board proceedings should promptly commence. Medical boards should be processed immediately, given priority over other boards. No more than seven working days should transpire between the date the board is dictated and the date of signature by the convening authority.

AMERICAN BOARD CERTIFICATIONS

American Board of Anesthesiology

CDR John D. Tolmie, MC USN
LCDR William B. Mahaffey, MC USNR

American Board of Internal Medicine

LCDR James L. Basiliere, MC USN
LCDR Charles S. Eytel, MC USN
LCDR Hugh S. Gallagher, MC USN
LCDR John J. Hand, MC USN
LCDR Julian Katz, MC USNR
LCDR Allan L. Mattern, MC USN
LCDR Kevin C. Stanton, MC USN

American Board of Neurological Surgery

CDR Victor G. Schorn, MC USN
CDR Israel D. Zuckerman, MC USNR
LT Richard E. Freeman, MC USN

American Board of Obstetrics and Gynecology

CDR Richard F. Johnson, MC USN
CDR Kelvin F. Kesler, MC USN
CDR John R. Miewald, MC USN
CDR Frederick J. Witt, MC USN
CDR Franklin M. Wolfe, MC USN
LCDR Julius L. Gray, MC USNR
LCDR Joseph L. Yon, Jr., MC USN

American Board of Ophthalmology

CAPT Lawrence M. King, Jr., MC USN
CDR David B. Davis, II, MC USN
CDR Richard J. Magenheimer, MC USN
CDR Robert H. Meaders, MC USN
CDR Lewis H. Seaton, MC USN
CDR James W. Smith, MC USN
LCDR Richard T. Torchia, MC USNR
LCDR Turpin H. Rose, MC USNR

American Board of Orthopaedic Surgery

CAPT David Q. Wilson, MC USN
CDR John W. Hauzenblass, MC USN
CDR Arthur H. Holmboe, MC USN
CDR Rodney I. MacDonald, MC USN
CDR Paul H. Randels, MC USN
CDR Guy B. Townsend, MC USN
LCDR Richard F. Ambur, MC USN
LCDR Jerry L. Case, MC USN
LCDR John B. Fenning, MC USN
LCDR M. Mirbaha, MC USNR
LCDR Norman L. Pollock, MC USNR
LCDR Albert G. Santarelli, MC USNR

American Board of Otolaryngology

LCDR Richard S. Crumrine, MC USNR
LCDR John A. Garrett, MC USN
LCDR Mario J. Guastello, MC USNR
LCDR Laurence E. Winter, MC USNR

American Board of Pathology

LCDR Thomas V. DiSilvio, MC USN
LCDR C. James Favino, MC USN
LCDR Robert J. Nelms, Jr., MC USN
LCDR Berton T. Schaeffer, MC USN

American Board of Pediatrics

LCDR William R. Hamilton, MC USN
LCDR Thomas M. Hanlon, Jr., MC USN
LCDR John D. Izsak, MC USNR

American Board of Plastic Surgery

CDR J. Vickers Brown, MC USN
LCDR Victor I. Rosenberg, MC USNR

American Board of Preventive Medicine

CDR Ralph D. Comer, MC USN

MEDICAL SERVICE CORPS EDUCATIONAL ACHIEVEMENT IN FISCAL YEAR 1969

For the past several years great effort has been expended by Medical Service Corps officers in their educational programs. Fiscal Year 1969 was a milestone in this area. The Medical Service Corps officers listed below were awarded academic degrees during Fiscal Year 1969 as indicated. It is interesting to note that many specialties are represented among the degrees which have been earned. BuMed Instructions 1500.7A and 1520.12D are applicable to the Medical Service Corps training program, and all officers are encouraged to take advantage of the financial opportunities available in furthering their education.

The Fiscal Year 1970 training program will show approximately the same number of academic degrees as were awarded in Fiscal Year 1969. While it is too early to accurately predict the Fiscal Year 1971 training input, it is anticipated that the same level of interest will be present as has been experienced during recent years.

Of the 48 degrees awarded to the below list of officers more than 95% were obtained through a considerable amount of part-time, off-duty efforts. Participation in part-time, off-duty educational programs continues to be a significant factor in the success of the Medical Service Corps training program. Approximately 225 Medical Service Corps officers are currently enrolled in part-time, off-duty courses during the 1969 Fall Semester. All officers are encouraged to consider their educational achievement in connection with their professional performance. BuMed will assist all officers in pursuit of their educational endeavor to the fullest extent possible consistent with the availability of training funds and billets.

Doctor of Philosophy

Degree	Degree	School	Duty Station
LT A. B. Cobet	Microbiology	Univ of N. H.	NMRU No. 1
LCDR R. E. Doll	Psychology	Univ of Utah	Navy Med Neuro-psychiatric Research Unit San Diego
LT R. D. McCullah	Psychology	Univ of Md.	NavDisp, TI, San Fran

Master's Degree

LT F. E. Bennett	Navy Mgmt	PGS, Monterey	Station Hosp, DaNang
LT E. Bobola	Navy Mgmt	PGS, Monterey	NavSupAct, Saigon
LCDR D. R. Craig*	Education	Univ of San Diego	NavDenCen, San Diego
LT J. W. Duley, Jr.	Nuclear Engr	Ga. Inst of Tech	NMS, NNMC, Bethesda
LCDR F. X. Faherty	Systems Mgmt (Automatic Data Processing)	GWU	Data Serv Cen. NNMC, Bethesda
CDR R. M. Garver	Physics	San Diego State Col	NH, St. Albans
CDR R. E. Meyer*	Education	GWU	Naval Academy, Annapolis
LCDR J. A. Nelson*	Financial Mgmt	GWU	AFIP, Wash, D. C.
LT A. E. Piatt*	Bus Adm	Boston Col	USS HORNET
LCDR J. J. Steil	Navy Mgmt	PGS, Monterey	USNH, Taipei
LT A. O. Woods	Health Care Adm	GWU	NH, Oakland

*Completed all Master's degree requirements entirely on a part-time, off-duty basis.

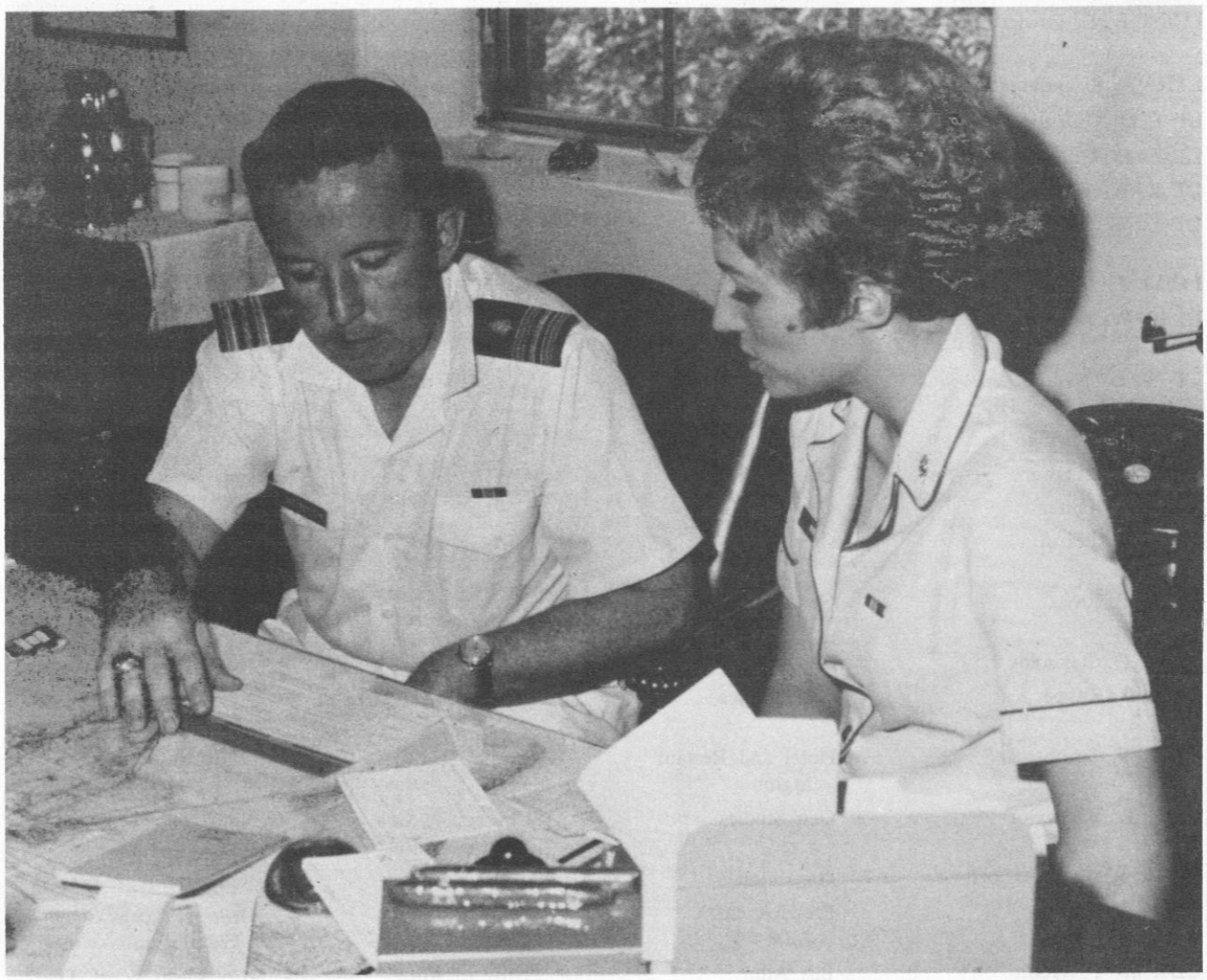
Baccalaureate Degree	Degree	School	Duty Station
LT A. T. Antonopoulos	Hotel and Restaur Mgmt	Cornell Univ	NH, Orlando
LT S. C. Bazzell	Gen Studies	GWU	USS REPOSE
LCDR R. R. Bowden	Gen Studies	GWU	NH, Camp Pendleton
LT F. W. Carter	Gen Studies	GWU	NH, Portsmouth, N.H.
LT D. E. Cassel	Gen Studies	Chaminade Col of Honolulu	U.S. Army Tripler General Hospital
LCDR H. T. Chastain	Gen Studies	GWU	BuMed, Code 36
LT N. J. Clem	Hotel and Restaur Mgmt	Cornell Univ	NH, Memphis
LT F. S. Conners	Gen Studies	GWU	Second MAW, FMFLANT
LT J. T. Dalton	Gen Studies	GWU	PGS, Monterey (DUINS)
LCDR K. L. Darr	Gen Studies	GWU	First Marine Div
LCDR B. R. Elfstrom	Gen Studies	Chaminade Col of Honolulu	NPMU No. 6
LCDR M. L. Fitts	Gen Studies	GWU	BuMed, Code 7A
LCDR E. E. Fowler	Gen Studies	GWU	NH, Portsmouth, Va.
LTJG R. S. Galbreath	Gen Studies	GWU	GWU (DUINS)
LT J. D. Gillentine	Social Sci	GWU	NMRU No. 3
LT F. D. Gillespie	Gen Studies	GWU	NMS, Bethesda
LT R. W. Horrobin	Social Sci	GWU	BuMed, Code 46
LT L. W. Johnson	Gen Studies	GWU	Scott Air Force Base
LCDR T. F. Levandowski	Social Sci	GWU	NMRU No. 2
LT T. W. MacConnell	Gen Studies	GWU	Data Serv Cen, NNMC, Bethesda
LT W. A. Nacrelli	Hotel and Restaur Mgmt	Cornell Univ	NH, Quantico
LT R. E. Newman	Gen Studies	GWU	BuPers
LCDR I. B. Owens	Gen Studies	GWU	NH, Pensacola
LT D. F. Potter	Gen Studies	GWU	GWU (DUINS)
LT P. A. Praria	Commerce	Univ of Md.	SubMedCen, New London
LT P. T. Ray	Gen Studies	GWU	NavDenClinic, Wash, D.C.
LT R. D. Reynolds	Gen Studies	GWU	NMRU No. 2
LT J. R. Ruppe	Gen Studies	GWU	NH, Portsmouth, Va.
LT R. E. Smith	Hotel and Restaur Mgmt	Cornell Univ	NH, Camp Pendleton
LTJG R. B. Taylor	Gen Studies	Chaminade Col of Honolulu	Naval Shipyard Pearl
LCDR R. L. Wentworth	Gen Studies	GWU	NavDisp, Wash, D.C.
LT J. A. Wilson	Pre-Hospital Adm	San Diego State Univ	Hospital Corps Sch, San Diego
CDR W. F. Woolf	Bus Adm	Univ of Md.	Nav Disp, Wash, D.C.

NAVY DIETETIC EXHIBIT

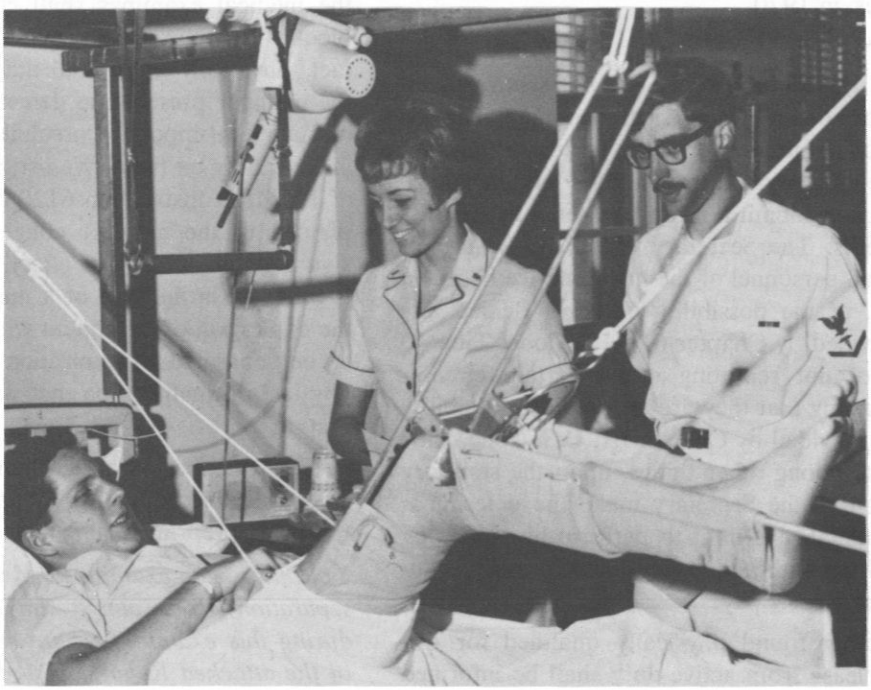
"Making Rounds With a Navy Dietitian" was the title of the Navy exhibit for the Fifth International Congress of Dietetics and the 52nd Annual Meeting of the American Dietetic Association held September 8th to 12th in Washington, D.C.

The exhibit illustrated the work of a Navy dietitian. It depicted the range of dietetic responsibilities and naval activities of LTJG Sandra Doppelheuer, MSC USNR, therapeutic dietitian at Bethesda Naval Hospital, Bethesda, Maryland. LTJG Doppelheuer also served as exhibit monitor, and began planning the make-up of the display in April. A Navy photographer followed in her footsteps attempting to capture the "Navy action" on film. The "action" began at Bethesda and included visits to the Naval hospitals at Annapolis and Patuxent River, Maryland, and Quantico, Virginia, where she consults on a monthly basis.

Over 6,000 dietitians participated in the International Congress. In addition to LTJG Doppelheuer, the following active duty Navy dietitians from hospitals around the world were present: LCDR Martha Springer, St. Albans, New York; LCDR Ernestine Phillips, Oakland, California; LCDR Patricia Hourihan, Yokosuka, Japan; LCDR Virginia Baker, San Diego, California; LTJG Beverly Green, Long Beach, California; LTJG Shirley Patterson, Portsmouth, Virginia; LTJG Charles Rath, Philadelphia, Pennsylvania; LTJG Nadine Neyer, Camp Lejeune, North Carolina; LTJG Wayne Singleton, Charleston, South Carolina and LT Michael Jenkins, Jacksonville, Florida. CDR Joan M. Beckwith, Assistant for Medical Specialist Officers in the Medical Service Corps Division of the Bureau of Medicine and Surgery, attended from Washington, D.C.



Some of the selected scenes included consultation with the doctor about the patient's needs and problems, instructing food service personnel on the aspects of a therapeutic diet, and the team approach (doctor, patient and dietitian).



NOTICES

The American Medical Association Section on Military Medicine elected a new slate of officers at the annual convention in New York City on July 14, 1969. CAPT Roger Stevenson, MC USN was elected Vice Chairman. CAPT Stevenson is currently assigned to the Bureau of Medicine and Surgery, and is Director of the Professional Division. Plans to attend the AMA Convention in Chicago in 1970 are already being formulated, and attendance at the scientific program of the Military Medicine Section is to be encouraged.

Military Medical Conference — CDR S. J. Kendra, MC USN attended the 5-day Central European Nations Treaty Organization Military Medical Conference, September 8–12. The conference this year was held in London, England; The Royal Air Force acted as host for the conferees. CDR Kendra, the Navy's representative, presented a paper, "The Control and Treatment of Meningococcal Meningitis." Among the principal benefits to be derived from conferences of this sort are better international communication in medical matters of mutual interest and more uniform methods of reporting the incidence of diseases. The Ninth Annual Conference is scheduled for an as yet, undesignated location in the United States, in 1970.

BUMED INSTRUCTION 6120.6D sets forth instructions for conducting physical examinations prior to separation from active duty, to insure that all members being separated from active duty are aware of their rights to submit a claim for Veterans Aministration benefits. The Secretary of the Navy has directed that all personnel of the naval service be made aware, also, of the possibility of being denied any benefits provided by Chapter 61, U.S. Code, Title 10 by reason of not rebutting under certain circumstances, a finding that they are fit for duty. Eligibility for benefits provided by Chapter 61, U.S. Code, Title 10, depends, among other things, upon the statutory requirement that the Secretary make the determination that a member is unfit to perform his duties by reason of physical disability while the member is entitled to receive basic pay.

Any member found physically qualified for discharge or release from active duty shall be informed by the examining medical officer that, subsequent to discharge or release from active duty, he cannot qualify for benefits provided by Chapter 61, U.S. Code, Title 10, for the reasons noted above. To insure that the member fully understands his rights in this area, the member shall be required to read the following:

You have been examined and found to be physically fit for separation from active duty. If you feel you have a serious defect or condition that is unfitting, tell the doctor who examined you. The doctor will evaluate your defect or condition and, if necessary, refer you to a hospital for further study and, if warranted by the further study, appearance before a medical board.

To receive a disability pension from the Navy, you must be found unfit to perform your duties before you are separated. After you are separated, any claims for disability benefits must be submitted to the Veterans Administration.

Please sign the statement on the back of the SF 88 (Report of Medical Examination), in block 73, to show that you understand the foregoing.

The member shall then be requested to sign the following entry in block 73 on the Standard Form 88: "I certify that I have been informed of and understand the provisions of BUMED Instruction 6120.6D."

Should a member refuse to sign the above entry, the medical examiner shall certify, by an entry in block 73 on the SF 88, that (1) the member is, in fact, physically fit for full duty and separation; (2) the member presents no defect or condition which is of sufficient import to preclude reasonable performance of duty on the active list; and (3) the provisions of BUMED Instruction 6120.6D have been fully explained to the member concerned who declines to sign a statement to that effect. (A similar procedure is followed in the case of a member retained beyond the expiration of enlistment when the member desires to be separated at expiration of enlistment despite physical disqualification that warrants further consideration.)

Each member separated from active duty shall be given a legible copy of his separation SF 88 with the following preprinted statement attached:

You have been examined and found physically fit for separation from active duty. Any defects noted during this examination are recorded in block #74 of the attached Report of Medical Examination (SF 88). Although the defects listed do not disqualify you for performance of your duties or entitle you to disability benefits from the naval service you may be

entitled to certain benefits from the Veterans Administration. In this connection, you should be counseled by the VA representative attached to your separation activity, if one is available, concerning the filing of claims for compensation with the Veterans Administration. Otherwise, it is suggested that you contact the VA Regional Office nearest your home as soon as practicable after your separation or retirement.

Based on unfavorable reactions resulting from the rental of television sets to servicemen hospitalized in naval hospitals, the Bureau has recommended in the past that an adequacy of television sets be insured and reasonable controls placed upon their issue and use. The Surgeon General has recently expressed his desire that hospitals use government-owned television sets to the greatest extent possible. Rental TV service is discouraged especially on those wards primarily composed of enlisted patients. On the other hand, the use of television sets owned by patients or furnished by their families should not be restricted, unless such use is medically contraindicated.

In the past year various hospitals have obtained a sufficient number of TV sets, either by purchase or donation, to obviate the need for rental services. Solicitation of TV rentals by concessionaires was thereby eliminated. At other hospitals rented sets were permitted only when specifically ordered by a patient in areas where no enlisted personnel were hospitalized.

Continued cooperation and effort concerning the above is appreciated.

Why Ice Dispensers: All hospital personnel are aware of the ice contamination problems—almost impossible to control when using standard ice machines.

Present ice machines all have storage bins, access to which requires opening a bin door. These doors are often left open, thereby exposing the ice to airborne bacteria. To remove the ice, a scoop is usually used that is impossible to keep aseptically clean, further contaminating the ice. Hands using the scoop, and other objects such as milk cartons, beverage bottles, lunches, etc. are also frequently introduced. Dispensers eliminate most of these problems.

Ice dispensers should be so designed that as the ice is made it goes directly into a sealed, insulated ice bin. When ice is required, one merely places a container, carafe, or bedside water pitcher under the spout permitting the ice to drop directly into the receptacle, dispensing the exact amount required. Opportunity for contamination is thereby minimized.

To further maintain the ultimate in cleanliness, all internal parts within the dispenser should be of stainless steel and except for the liner should be removable without use of tools for sanitizing. All parts being of stainless steel may be autoclaved if desired.

Meeting of Military Surgeons

The Association of Military Surgeons of the U.S. will hold their 76th Annual Meeting at the Sheraton Park Hotel in Washington, D.C. on November 17-19, 1970. Guest badges will be issued at the Registration Desk.

Dr. Gerald D. Dorman, President of the AMA, will give the keynote address on Nov 17, "Teaming Up for Quality Health Care."

Most of the professional papers will be given at panel discussions where care of patients suffering from disease, shock and trauma will be among the subjects discussed by top authorities.

Some 115 technical and scientific exhibits will be displayed and prize-winning films will be shown each day.

An excellent program has been set up for wives of visiting delegates, including a visit to the White House with Mrs. Nixon receiving the group.

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